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TYPHOID FEVER.

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THE MEDICAL COMPLICATIONS, ACCIDENTS AND SEQUELÆ

OF

TYPHOID OR ENTERIC FEVER.

BY

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WITH A SPECIAL CHAPTER ON THE

MENTAL DISTURBANCES FOLLOWING TYPHOID FEVER.

BY

F. X. DERCUM, M.D.,

CLINICAL PROFESSOR OF DISEASES OF THE NERVOUS SYSTEM IN THE JEFFERSON MEDICAL COLLEGE.

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PREFACE.

At the present time there are few diseases so widespread as typhoid fever, and the literature concerning it is very great. Systems of medicine and text-books innumerable deal with its ordinary manifestations, and touch, necessarily but briefly, upon its accidents, its complications, and its sequelæ. Anyone who has had even a limited experience with typhoid fever has met with cases in which the manifestations wandered so far from the classical descriptions of the disease as to be puzzling and obscure, or with instances in which the malady has been so altered in its course by intercurrent affections as to be unusual and to call forth all the diagnostic knowledge and therapeutic skill of the physician. The following pages deal with these aberrant forms of the disease and the courses which they pursue.

As mental disorders sometimes complicate typhoid fever, I have asked my colleague, Dr. Dercum, to add a chapter on this phase of the subject, which is of great interest.

Finally, I desire to acknowledge my great indebtedness to the several authors who have enriched medical literature by special contributions to this subject, and from whose writings and bibliographical researches I have gained much valuable material. The first of these is the essay of my honored colleague, Dr. W. W. Keen, on the *Surgical Complications and Sequelæ of Typhoid Fever*. In many instances Dr. Keen, in completing his statistics, steps into the bounds of medicine, in distinc-

tion from surgery, and in this way our studies sometimes overlap. Another writer to whom all subsequent authors on typhoid fever are indebted is Liebermeister, whose classic article in Ziemssen's *Encyclopœdia* is well known. I am also anxious to acknowledge my indebtedness to the writings of Osler, Mason, and Fitz.

222 South Fifteenth St., Philadelphia,
April, 1899.

CONTENTS.

CHAPTER I.

General Considerations 17

CHAPTER II.

Varieties of Onset 37

CHAPTER III.

The Aberrant Symptoms, States, or Complications of the Well-developed Stage of the Disease 63

CHAPTER IV.

The Complications of the Period of Convalescence . . . 174

CHAPTER V.

The Conditions which Ape Typhoid Fever 253

CHAPTER VI.

Duration and Immunity to Second Attacks 262

CHAPTER VII.

The Mental Complications 265

THE MEDICAL COMPLICATIONS AND SEQUELÆ OF TYPHOID OR ENTERIC FEVER.

CHAPTER I.

GENERAL CONSIDERATIONS.

It may be said by those who are disposed to be critical, that an essay dealing with the medical complications and sequelæ of typhoid fever must of necessity deal with the disease in so wide and general a manner as to include practically all that we know concerning it; but, while this is to a certain extent true, on the other hand, it is manifest that the important subjects of etiology and pathology will not find space for their consideration, and that the simple unaltered forms of the malady will only have to be described sufficiently to indicate the real variations. No one who has had any experience with this disease can fail to have noted that it presents widely different symptoms in degree and in kind, not only in different epidemics, but in different individuals, and in the same individual at different periods of a single attack. In some patients the illness is so mild as to be only a moderate indisposition; in others so malignant that death speedily ensues, and yet in nearly all cases there are certain manifestations which when grouped together render it possible to make a diagnosis fairly certain. A febrile course, characterized by malaise, headache, fever, drowsiness, intestinal disorder, enlargement of the spleen and liver, the eruption of rose spots, and the confirmatory Widal test, may be considered to represent true uncomplicated typhoid fever; and with cases presenting these general symptoms this essay will not deal. On the other hand, the object in view is to discuss three classes of

the manifestations of typhoid infection, namely, (*a*) those ordinary symptoms of onset and complete development which, by reason of moderation or modification or exaggeration, become interesting or dangerous in themselves; (*b*) those which are so rarely met with during onset or the course of the malady in ordinary cases that they can be considered as distinctly complicating conditions, and (*c*) those results of the disease which, coming on after it is about to cease in itself, still retard or interfere with the rapid and normal return of the patient to perfect health.

I am well aware that at certain points it will seem that the dividing line between the ordinary symptoms and those considered in these pages is overstepped, and while it is not my intention to avoid this overstepping when the complete discussion of the condition is necessary to a thorough study of the process under consideration, these ordinary symptoms will not, as a rule, be generally considered.

Before proceeding to a clinical study of the disease, it is interesting to note that its frequency, severity, and mortality are distinctly on the wane. While isolated epidemics may range in severity from mild to severe, and produce a mortality from less than 1 per cent. to almost 50 per cent., the average being at one time about 25 per cent., the mortality is now much less than this, and often only 10 per cent., and in private houses where the family is well enough placed to give the patient every aid, it is often less than 5 per cent., even when the treatment instituted is not all that could be desired.

These changes have been produced by improved sanitation, a natural modification in the severity of the infection, coupled, perhaps, with an increased resistance on the part of the individual, and by better treatment, and as they bear an interesting relation to other modifications of the malady, may be discussed at this point with propriety. In regard to the effect of improved sanitation it can be pointed out that Mosny has shown that the death-rate of Vienna decreased from 12.05 per 10,000 to 1.1 after a pure water-supply. In Dantzic the mortality has fallen from 10 per 10,000 to 2.4, and finally to 1.5 per 10,000. In Stockholm it fell from

5.1 in 1877 to 1.7 in 1887. So, too, in Boston from 17.4 in 1846–49 to 5.6 in 1870–84.

The following table is of interest in this connection:

MORTALITY IN MUNICH FROM 1851 TO 1896.

Year.	Inhabitants.	Annual.	Per 100,000 inhabit'nts.	Year.	Inhabitants.	Annual.	Per 100,000 inhabit'nts.
1851,	123,957	123	99.0	1874,	181,300	289	159.0
1852,	125,588	152	121.0	1875,	187,200	227	121.0
1853,	127,219	235	184.0	1876,	193,024	130	67.0
1854,	128,850	293	227.0	1877,	205,000	173	84.0
1855,	130,481	253	193.0	1878,	211,300	116	55.0
1856,	132,112	384	291.0	1879,	217,400	236	109.0
1857,	133,847	390	291.0	1880,	223,700	160	72.0
1858,	135,733	453	334.0	1881,	230,028	41	18.0
1859,	137,005	240	175.0	1882,	236,400	42	18.0
1860,	140,624	153	109.0	1883,	242,800	45	19.0
1861,	144,334	172	119.0	1884,	249,200	34	14.0
1862,	148,200	300	202.0	1885,	255,600	45	18.0
1863,	154,602	252	163.0	1886,	262,000	55	21.0
1864,	160,828	397	247.0	1887,	268,400	28	10.0
1865,	167,054	338	202.0	1888,[1]	292,800	31	10.5
1866,	168,265	342	203.0	1889,	306,000	31	10.1
1867,	169,476	88	52.0	1890,	331,000	28	8.5
1868,	170,688	136	80.0	1891,	357,000	24	6.4
1869,	170,000	190	111.0	1892,	372,000	11	3.0
1870,	170,000	254	149.0	1893,	385,000	57	14.8
1871,	170,000	220	129.0	1894,	393,000	10	2.5
1872,	169,693	407	240.0	1895,	400,000	15	3.7
1873,	175,500	230	131.1	1896,	412,000	14	3.4

The effect of improved sanitation is to decrease the virulency of infection, and for this reason there follows a decreased severity of illness and a decreased percentage of mortality. Not only are these facts true of the cities just named, but it is also true that the frequency, severity, and mortality of typhoid fever are steadily decreasing all over the world, as is shown by the following interesting tables of Dreschfeld in regard to England in general and London and Manchester in particular:

[1] This table is taken from Pettenkofer's "Munich a Healthy City," up to 1887 inclusive; after 1887 from returns obtained from the Statistical Bureau.

ANNUAL MORTALITY, PER MILLION PERSONS LIVING, FROM FEVER IN ENGLAND.

Period.	Enteric cases.	Period.	Enteric cases.
1838	1228	1866	986
1839	1010	1867	778
1840	1089	1868	895
1841	932	1869	390
1842	1004	1870	388
1843	.....	1871	371
1844	.....	1872	377
1845	.....	1873	376
1846	.....	1874	374
1847	1807	1875	371
1848	1266	1876	309
1849	1044	1877	279
1850	865	1878	306
1851	997	1879	231
1852	1022	1880	261
1853	1008	1881	212
1854	1015	1882	229
1855	875	1883	228
1856	847	1884	236
1857	988	1885	175
1858	918	1886	184
1859	806	1887	185
1860	652	1888	172
1861	767	1889	176
1862	919	1890	179
1863	874	1891	168
1864	960	1892	137
1865	1089		

DEATH-RATE FROM ENTERIC FEVER IN LONDON AND MANCHESTER PER MILLION.

Year.	London.	Manchester.	Year.	London.	Manchester.
1871	267	450	1883	247	200
1872	242	400	1884	234	190
1873	269	460	1885	150	170
1874	256	390	1886	154	290
1875	235	440	1887	151	310
1876	217	420	1888	169	330
1877	251	290	1889	130	310
1878	283	310	1890	146	270
1879	229	180	1891	132	370
1880	186	260	1892	102	240
1881	254	170	1893	161	250
1882	252	250			

These figures are exhibited graphically in the following chart:

Fig. 1.

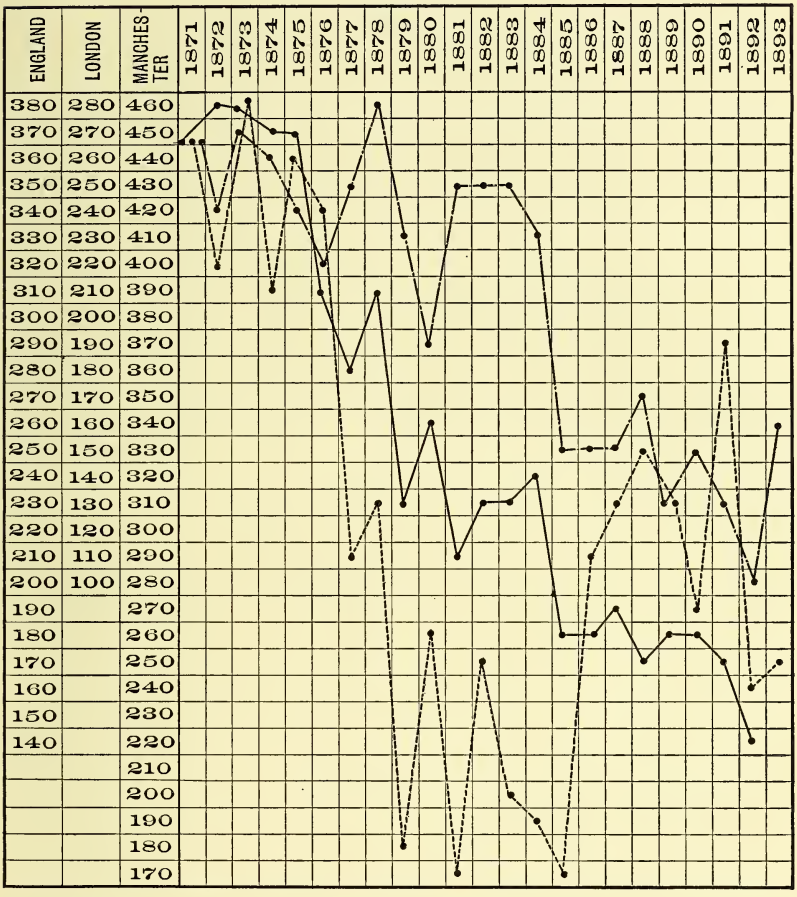


Chart showing decreasing mortality of typhoid fever per million persons living in England, London and Manchester.

Solid line, England. Broken line, London. Dotted line, Manchester.

Not only is the decrease in mortality seen in England, but in Philadelphia and New York, as follows. The decrease in cases and in mortality in Philadelphia is shown in the following chart in broken and complete lines (Fig. 2):

FIG. 2.

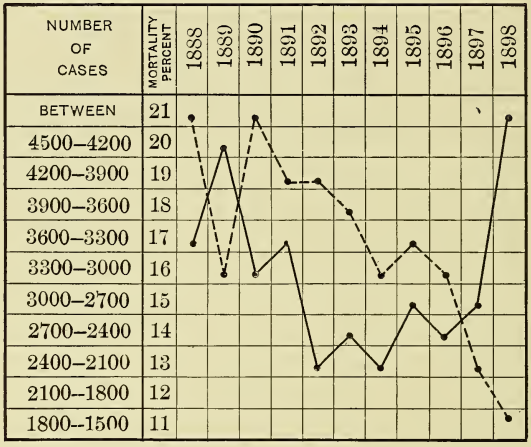


Chart showing the morbidity and mortality of typhoid fever in Philadelphia. Notwithstanding the present epidemic which in 1898 raised the morbidity from between 2700 and 3000 to over 4500, it will be seen from the dotted line that the mortality per cent. still decreased.

Solid line, morbidity. Dotted line, mortality.

PHILADELPHIA.

Year.	Cases.	Deaths.	Per cent. of mortality.
1888	3573	785	21.9
1889	4631	736	15.8
1890	3182	566	20.9
1891	3531	683	19.3
1892	2304	440	19.1
1893	2519	456	18.1
1894	2357	370	15.7
1895	2748	469	17.0
1886	2490	402	16.1
1897	2994	401	13.3
1898	4749	566	11.91

These statistics go back as far as the comparative records extend, and do not include the 1348 soldiers with typhoid fever who returned from the Spanish-American war in 1898, but only the regular population of the city. If the soldiers are added, to the number of 1348, we find that 6097 cases of enteric fever occurred in Philadelphia in 1898. The mortality of the city population was 11.91, that of the soldiers 5.41, which would

make the total percentage 10.47 in 6097 cases. The low mortality of the soldiers is a tribute to hospital treatment, for in many cases these men were transported hundreds of miles when very ill, and, as a rule, had not had the food and care which are so necessary to the safe conduct of a typhoid case. Again, while the frequency of the disease has risen from 2994 cases in 1897 to 4749 cases in 1898, the mortality is only 11.91 for 1898, or, if the soldiers are included, making 6097 cases, 10.47 per cent.

Fig. 3.

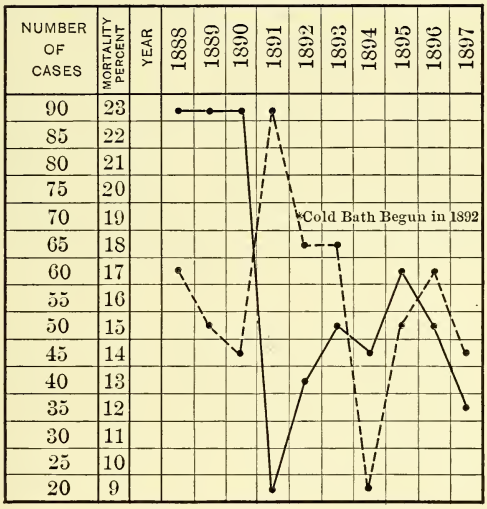


Chart showing morbidity and mortality per cent. at the Philadelphia Hospital for ten years (1888–1897 inclusive). Both the morbidity and mortality are decreased.

Solid line, morbidity. Dotted line, mortality.

As only a little over two months of 1899 have elapsed the statistics for this year cannot be included in Fig. 2; but it is interesting to note that, while this wide-spread epidemic, due to bad water, has persisted and increased, *the mortality per cent. has not increased.* Thus in 1899, up to March 13th, no less than 3424 cases of typhoid fever occurred of which 360 cases died, or 10.51 per cent.

FIG. 4.

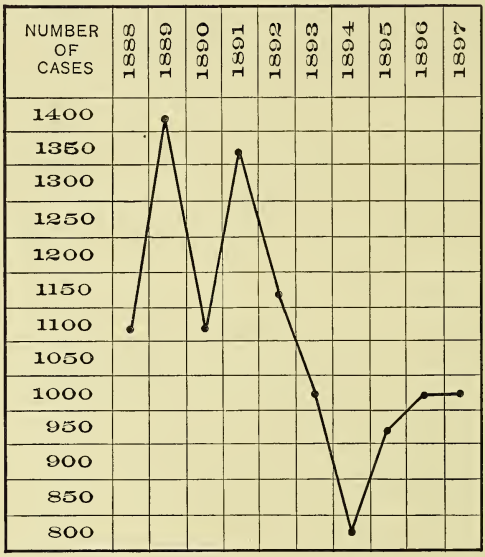


Chart showing decreasing number of cases annually in New York.[1]

FIG. 5.

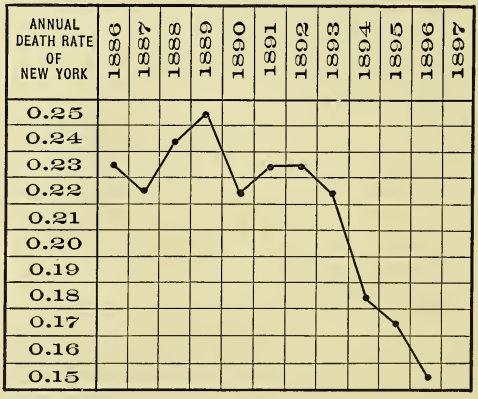


Chart showing decreasing death-rate from typhoid fever in New York City.

[1] A comparative chart of the number of cases and of mortality per cent. from these figures is not given, as Dr. Biggs, of the New York Health Office, writes that only recently have the cases been generally reported, and even now many are not reported.

New York.

Year.	Cases.	Deaths.	Year.	Cases.	Deaths.
1888	1108	364	1893	1008	381
1889	1414	397	1894	792	326
1890	1100	352	1895	965	322
1891	1342	384	1896	1002	297
1892	1140	400	1897	1004	299

The chart on preceding page from the New York Health Report shows a decrease in death-rate from typhoid fever (Fig. 5):

When we consider that the population of these cities has increased enormously, the great decrease in the frequency of the disease and in its mortality is very notable.

These tables are supported by the statement of Billings, that in Norway from 1888 to 1891 the mortality from typhoid fever was 755 in 7467 cases, or less than 10 per cent. In the recent Maidstone epidemic the death-rate in 1885 cases was only 7.5 per cent., and a similar mortality obtained at Plymouth, Pa. The death rate in the Worthing epidemic of about 1000 cases was 13 per cent.

Bryant[1] states that out of 608 cases treated in Guy's Hospital from 1879 to 1893 14 per cent. died.

Again, in the *Gazette Médicale des Hôpitaux* of July 10,1890, we learn that a collective investigation showed that, whereas in the period from 1866 to 1881 the mortality from typhoid was 21.5 per cent.; from 1882 to 1888 it was 14.1 per cent., and in 1889, 13.5 per cent.

We may assume then that the ordinary mortality of typhoid fever is at present less than 15 per cent. in the general run of cases, and that in good hospitals and private practice with good nursing, that it varies from 1 to 10 per cent., the more so as many years ago, before the disease had become modified, Murchison placed it at 17.45 among 27,951 cases in England.

The following statistics of patients treated by general methods show this to be true, and with or without baths a similar decrease in mortality is evident:

[1] Guy's Hospital Reports, 1893.

	Cases.	Per cent, of mortality.	Treatment.
Basel (Liebermeister)	223	11.7	Calomel.
Basel (Liebermeister)	239	14.6	Iodide.
Maidstone, England	1885	7.5	General.
Boston (Mason)	676	10.4	General.
Homerton (Collie)	677	9.5	General.
Glasgow (Collie)	618	8.2	General.
Société Médicale des Hôpitaux (1879)[1]	1979	12.47	
Jaccoud	665	10.8	General.
Riess	900	7.5	Tepid baths.
Boston (Shattuck)	237	9.8	Expectantly and cold sponging.
Germany (?) Brand has collected	19,017	7.8	All kinds of cold baths.
	27,116	10.02	

In other words, 27,116 cases in Switzerland, America, England, Germany, and France show that good nursing and careful non-meddlesome treatment will give a mortality of about 10 per cent. The wide distribution of these cases and the large number of clinicians give us a standard average.

At Basel in 1873, under the cold bath, there were 163 cases, with a mortality of 10.4 per cent.; during the same year at Glasgow without baths, 275 cases, with a mortality of 9.4 per cent.; and 305 at Homerton, with a mortality of 9.5 per cent. In 1874 at Basel the water cases were 200, with a mortality of 10.5 per cent.; at Homerton 372, with a mortality of 9.6 per cent.; at Glasgow 343, with a mortality of 7 per cent.

	No. of cases.	Treatment.	Mortality per ct.
Basel (1873)	163	Bath	10.4
Glasgow "	275	General	9.4
Homerton "	305	General	9.5
Basel (1874)	200	Bath	10.5
Glasgow "	343	General	7.0
Homerton "	372	General	9.6

[1] These statistics are based upon the fact that twenty-one chiefs of hospital service reported to the Société Médicale des Hôpitaux (1890) 916 cases with 114 deaths, or 12.44 per cent. under general treatment; and for 1888 and 1889 this report also mentions 1063 cases so treated with 133 deaths, or 12.51 per cent.

Of the fact that a change in type has taken place in enteric fever, I do not think there can be any doubt, and no one who has watched the disease during the last fifteen or twenty years, or even for a shorter period than this, can fail to note the difference in its character. Particular attention has been called to this fact by Sidney Phillips[1] and James F. Goodhart.[2] The latter writer says: "I agree *in toto* with what Dr. Sidney Phillips said to us that 'typhoid fever tends to vary with the conditions associated with its origin, and though such variations are slight individually and gradual in evidence in their sum, they suffice in time to produce a considerable modification of the original disease. There is considerable difference in the symptoms described fifty or even twenty-five years ago and those occurring to-day. The difference is marked in the lessened severity of the abdominal symptoms; the tongue is now often moist throughout the disease, instead of dry and baked; tympanites and diarrhœa are much less pronounced; probably also hemorrhage and perforation are less common; tremors and dilatation of the pupils are now uncommon; and, instead of noisy, active delirium, the mind is often clear throughout even fatal cases. The typhoid state with the patient sunk deep in bed, unable to move himself and unconscious or semi-conscious for days, is now quite exceptional. Dr. Phillips attributes this 'to a lessened tendency to ulceration of the intestines,' and argues that if so much variation of type has taken place in a quarter of a century, much more has gone on in fifty years, and that where conditions existed such as made typhus rife the distinctive features of typhoid may well have been affected, and that in this is possibly to be found the explanation that the separate diseases were regarded as one."

In this connection the question of the *frequency of typhoid fever in children* may be considered. At first sight it would appear that in this class of patients it is a more common disease than formerly, but this is only because it was not recognized and recorded.

[1] British Medical Journal, November, 12, 1898. [2] Ibid., January 28, 1899.

Typhoid fever in children is by no means as rare as has been supposed. While the earlier years of life seem to be blessed with a relative immunity to the disease, there is no doubt that it often occurs in a mild form and is not correctly diagnosed. A young child sickens, has fever, is wretched, has moderate diarrhœa or constipation, and a coated tongue. Debility is rapidly developed, the stomach becomes irritable, and the fever is persistent, even though it is not high. After an illness lasting for from a few days to several weeks, the child gradually recovers, and the diagnosis originally made is adhered to, namely, that the case has been one of "simple catarrhal fever." The longer one practices medicine the more strongly the idea develops that such a thing as "simple catarrhal fever," does not exist as an entity, and that this term covers a multitude of diagnostic sins. As was pointed by Liebermeister years ago, typhoid fever may occur even in adults with these mild symptoms, and be called "catarrhal fever."

It may be laid down, however, as a rule, that the younger the child the less likely is it to have enteric fever, and that the prognosis is usually favorable if the child be young. In other words, the older the child, the more grave the prognosis. On the other hand, it is only fair to state that Rocaz[1] believes that while the duration of the fever in children is shorter than in adults, the fever itself is apt to be excessive; that the prognosis is grave under three years, less grave at four years, and only less grave than in adults when the child is above five years of age.

This question of how frequently typhoid fever does occur in children is of great importance. At the head of those who advocate the view that it is common we have Ashley and Wright,[2] who assert that "children and young people are more susceptible to typhoid fever than are adults, though it is not common in children under three years of age." This is certainly an excessive statement, although Pepper[3] states that typhoid fever is far more common in early life than is generally recognized. Henoch records

[1] Annales de la Polyclinique de Bordeaux, 1897.

[2] Diseases of Children.

[3] American System of Medicine, vol. ii.

376 cases and 26 autopsies in children from this disease, and Barthez and Sanne state that the disease is as frequent among children as among adults.

On the other hand, there is an immense amount of evidence to prove that the disease is so rare as to be almost a curiosity in children. Thus William Perry Northrup has taken the statistics of the New York Foundling Hospital, the New York Infant Asylum, the Children's Hospital of Philadelphia, and finds that in the twenty years at the New York Foundling Hospital with 1800 cases under care, 1100 of which were boarded in the country, returning to the hospital when ill, not a single case has been seen by himself, J. Lewis Smith and O'Dwyer. Further, in 2000 autopsies on children Northrup did not find a case, perhaps because typhoid fever rarely brings a child to autopsy, and during an epidemic in Stamford, Conn., in 1895, out of 400 cases at all ages, but four cases of enteric fever developed under four years of age.

Holt[1] states that he has never met with enteric fever in a child under two years of age. He never saw a case in the New York Infant Asylum in a service of eight years, although 15,000 cases were admitted in that time.[2] One case was admitted to the Babies' Hospital in seven years at the age of two and one-half years.

In this connection it is interesting to note that Taupin,[3] writing sixty years ago, says that the rarity of this fever in children is more apparent than real, and points out that the mild manifestations of the disease are overlooked.

Notwithstanding these statistics, we find that typhoid fever does occur quite frequently in the hands of some practitioners. Thus Forchheimer[4] treated 70 cases in 1888 in one epidemic, and Morse, in analyzing 284 cases in the Boston City Hospital in which this disease appeared, found 3 under five years of age, 77 between five and ten years, and 204 between ten and fifteen years.

[1] Diseases of Children.

[2] Probably all these did not come under his term of service.

[3] Journal des Connaissances Méd. and Chir., 1839, No. 7.

[4] American Lancet, March, 1889.

Holt quotes 970 cases of enteric fever in children collected from eight authors whose names he does not give. Of these 970 cases, 8 per cent. occurred under five years; 42 per cent. between five and ten years, and 50 per cent. between ten and fifteen years. He also quotes an epidemic of 115 persons, of whom three were under two years of age.

Wightman[1] has recorded 24 cases of typhoid fever in children under thirteen years of age; 3 of these died, and typical spots were seen in 15; constipation in 10, and typical stools in only 3 cases. So, too, Davis[2] has recorded 33 cases in children, all of whom were under ten years of age, and in all of whom but 3 the disease developed abruptly.

Ssokolow,[3] in a study of 581 cases of typhoid fever, the majority of which occurred between four and ten years, found that it was abortive in 4.3 per cent., mild in 26 per cent., ordinary in 51 per cent., and severe in 18 per cent. In 3.6 per cent. there was an abrupt onset with vomiting, and in 3.2 per cent. it was abrupt with a chill; diarrhœa occurred in only 10 per cent.

Bridges has met with the disease in infants at fifteen and eighteen months of age, and Bond saw eleven children, affected in one-house epidemic, between the ages of three and twelve years. Read[4] has collected 22 cases between four and one-half months and ten years, and Griffith reports cases at three, seven, eleven, and thirteen years. England records one at eight months of age, and Boobbyer one in an infant of eight months. Murchison recorded one at six months. Ogle has recorded a case at four and one-half months, and Fuller one at five months.

Further than this, Dr. Mart,[5] of German, Ohio, has recorded the fact that in six years he had treated seventeen cases of unquestionable typhoid in children ranging from fourteen months to five years of age; that three of these cases were less than twenty-four

[1] British Medical Journal, May 5, 1894.
[2] Alabama Medical and Surgical Age, August, 1894.
[3] Centralblatt für innere Med., May 18, 1895.
[4] Brooklyn Medical Journal, October, 1890.
[5] Cleveland Medical Gazette, vol. xii. p. 510.

months old, and in each instance there were other members of the family sick with the fever at the same time, showing that the infection was present in the household.

H. J. Lee,[1] of Cleveland, reports a case of typhoid fever in an infant six months old, and states, although he does not give the reference for the same, that he finds one case reported as young as four and one-half months;[2] another at six months, and a good many under two years.

O'Malley[3] records three cases of typhoid fever at twenty-one months, three years, and six years in one family.

Not only may typhoid fever occur in very young children, but it is to be remembered that this source of infection may cause the disease among adults. Thus Boobbyer[4] records an instance in which out of a family of eight persons five became infected through an infant of eight months. The child had been restless and had constant diarrhœa, but the fact that it was suffering from typhoid fever was not recognized.

That severe typhoid fever may occur very early in life is shown by the statement of Osler, that perforation of the bowel from this cause has occurred in a child five days old, and Earle has reported a case to Keating of fatal intestinal hemorrhage due to typhoid fever at twenty-two months.

Further than this, Sbrana,[5] who has treated seventy-two cases of typhoid fever in children in Tunis, tells us that a symptom which was never lacking was splenomegaly appreciable from the fifth or sixth day of the fever. The nervous symptoms were more marked in girls than in boys. The mortality was 11.1 per cent., and the complications were meningitis, suppuration, parotiditis, peritonitis from perforation, purulent pleurisy, aphasia lasting as long as three weeks, dilatation of the stomach during convalescence, and orchitis.

[1] Cleveland Journal of Medicine, 1897, vol. ii. p. 400.

[2] Probably Ogle's case. [3] University Medical Magazine, 1896–97, p. 637.

[4] British Medical Journal, January 26, 1890.

[5] Quoted in the American Journal of Obstetrics for March, 1899, from the Archives de Méd. des Enfance, January, 1899.

Wurtz[1] records the case of a girl of eight years, who developed a swelling over the sternum during the second week of typhoid fever. Puncture drew pus and an incision gave exit to a necrosed piece of the sternum, the entire body of the bone being involved in the necrotic process. Typhoid bacilli were demonstrated microscopically in the pus. Death occurred in the fifth week. At the autopsy an abscess lined by pyogenic membrane was found between the sternum and pleura, extending upward to the manubrium. There was broncho-pneumonia in both lungs, and in the ileum there were a few typhoid ulcers, the rest of the intestine showing healing; the right arytenoid cartilage showed a chondritis.

In the Maidstone[2] epidemic of 1897 and 1898, 22 per cent. of the cases admitted to the hospital were in children under ten years of age, and 52 per cent. were under fifteen years.

I think it is fair to conclude therefore that Taupin's assertion, in 1839, that typhoid fever is not a rare disease in children is correct.

At the present time the diagnosis of typhoid fever in children must rest largely upon the chance development of the characteristic rash and enlarged spleen, and more than all upon the Widal test, for the moderation in all the symptoms so characteristic of the affection in childhood, and the fact that a swollen spleen and liver and a coated tongue with fever are so commonly met with in various children's ailments, make an absolute diagnosis without this test in many instances almost impossible.

Typhoid fever *is not common in pregnancy*, but when it occurs it is a serious matter, for abortion often follows, particularly if the fever be high. The percentage of abortion is about 56 per cent. In 310 cases collected by Sacquin, 199 aborted. The mortality, according to Brieger, was 19 in 91 cases, and according to Vinay, 17 per cent. in 183 cases.

Death to the fœtus does not always occur as a result of prema-

[1] Quoted in the American Journal of Obstetrics for March, 1899, from the Jahrbuch f. Kinderheilkunde, vol. xliv., No. 1. I have not been able to see the original article.

[2] Poole. Guy's Hospital Reports, 1898. Wrongly labelled on cover 1896.

ture birth due to typhoid fever; thus Touvenaint[1] reports a case of premature birth at the end of the seventh month, the child surviving and the mother dying.

Typhoid fever may also affect the fœtus *in utero*. This Fordyce has proved, and he also asserts, that the child may survive. It is possible, too, for it to escape the infection. Flexner has examined such a case for Osler.

Griffith found the Widal reaction in a child of seven weeks whose mother had typhoid fever at the time of its birth.

So, too, Étienne[2] has recorded the examination of a fœtus expelled by a woman in the fifth month of pregnancy, on the twenty-ninth day of typhoid. The spleen and intestines of the child showed no signs of the disease, and the placenta was healthy, but an examination of the blood in the right side of the heart and of that of the spleen revealed innumerable typhoid bacilli.

Mossé and Daunic also record a case in which a woman suffered from typhoid fever in the eighth month of pregnancy. At birth the blood of the child, the blood of the placenta, and the milk of the mother gave the Widal reaction, as did the child thirty-three days after birth.

Another interesting illustration of the fact that the fœtus may become infected by the typhoid bacillus through the mother is shown by a case reported by Eberth,[3] of a woman who suffered from typhoid fever in the fifth month of pregnancy and miscarried, and in the cardiac and splenic blood of the fœtus the specific bacillus was found.

Mossé and Fraenkel[4] have made a report upon the agglutination test in placental blood to the Société Médicale des Hôpitaux, in which they confirm the statements already made, that the Widal test can be obtained from the placenta, and also that it is possible to obtain it from the milk of the mother and the blood of the fœtus.

[1] Journal de Médicine de Paris, July 8, 1894.

[2] Gazette Hebd. de Médecine et de Chirurgie, 1896, No. 16.

[3] Centralblatt für Bakteriologie and Parasitenkunde, May 13, 1890.

[4] Journal des Practiciens, January 28, 1889.

The following very interesting case in this connection has just been reported to me by my friend, Dr. Wilmer Krusen :

Mrs. B., aged twenty-seven years, a native of Ireland, a housewife by occupation, was admitted to the hospital February 7, 1899, eight months pregnant. From her attending physician it was learned that for a week prior to her admission she had had a typical typhoid temperature and stools, but no spots. On admission her temperature was 100.5°; the pulse was 100 ; respirations, 24. The temperature fell steadily till it reached 95° at 10 A.M. of February 8th, remaining there all that day ; the pulse ranging between 80 and 94, and the respirations between 18 and 32. About 1 A.M., February 8th, she developed labor pains, which lasted until 3 A.M., when they ceased entirely ; the pains were never severe, and labor progressed very slowly. The temperature was subnormal all the time, but began to rise toward morning, and reached 99° at 8 A.M.; the pulse, 100 ; respirations, 36. The temperature continued to rise slowly. At noon on February 9th very mild labor pains again began, but soon ceased. At 3 P.M. the child's head had descended entirely without any pain whatever. No progress being made, forceps was applied and the child delivered a few minutes past 3 P.M. Temperature, 100.4°; pulse, 136 ; respirations, 36. Temperature then went up, and at 6 P.M. was 103.4°, and continued with daily remissions, as is usual in typhoid. A superficial median laceration occurred ; it was sewed up, but no healing process took place, and the stitches had to be removed. About the tenth day after admission, the temperature became very irregular, ranging from 97° to 106.2°; pulse from 110 to 150 ; respirations, 20 to 44. The vaginal discharge had been copious and offensive, and continued so until the twenty-second day in the hospital. The temperature continued to be irregular throughout the remainder of the disease. From February 18th to February 24th the temperature became reversed, so that it was highest about 6 A.M. and lowest about 6 P.M., being still very irregular. From February 24th the temperature again assumed its former character, highest in the evening and lowest in the morning. The vaginal discharge had completely stopped by February 28th, having

been very slight for the preceding three or four days. March 3d, the temperature was 98.4° at 10 A.M. ; pulse, 92 ; respirations, 24. The child progressed nicely. The Widal reaction was taken March 2d with a very high dilution, and proved to be negative, though there was a distinct tendency to agglutination. It was taken again March 4th with a dilution of 1 part of serum to about 25 parts of water. The result was a positive reaction in eleven minutes.

A somewhat similar case has also been recorded by Batty Shaw.[1] A woman suffering from typhoid fever in the fifth month of pregnancy and her child gave a feeble Widal test five weeks after birth on two occasions, but on two other occasions the test was negative.

Two cases illustrating typhoid infection during the last weeks of pregnancy have recently come under my care, having been transferred to me from the Jefferson Maternity Wards by Dr. E. P. Davis. In both of them the fever began practically simultaneously with parturition, indicating that the patient had become infected during the last two weeks of pregnancy. In neither one of them were the typhoid manifestations severe so far as nervous and circulatory symptoms were concerned, but in one the temperature was fairly high and persistent. The blood of the children did not give the Widal test.

In patients over forty years, typhoid fever is a rare but grave disease, the mortality increasing with the years. The fever, as already indicated, is apt to be mild, but death comes more commonly than in comparative youth from complications like pneumonia and heart lesions (Fig. 6).

Dreschfeld has reported a case of typical typhoid fever in a man of seventy-five years, and another in a man of eighty-two years. In the latter case he states that recovery took place.

While it is generally true that the period of incubation of typhoid fever extends over a period from ten days to two weeks, recent reports indicate that in certain instances this period may only cover a few days. Thus Janehen-Graz[2] has recently reported thirty-six cases of typhoid fever occurring among soldiers, in whom

[1] London Lancet, 1897, vol. ii. p. 539.

[2] Münchener Medicinische Wochenschrift, 1898, p. 936.

definite proof was adduced that they had all become infected at the same time by drinking infected water. As a result the incu-

Fig. 6.

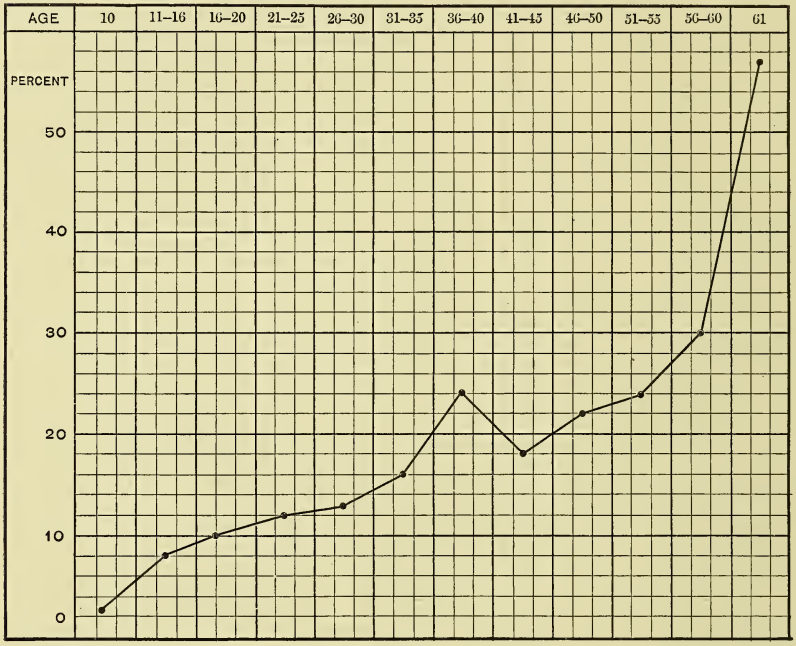


Chart showing the increasing mortality of typhoid with advancing years. (Curschman.)

bation period in three cases was only two days, in seven cases three days, in six cases four days, and in thirteen cases five to seven days.

CHAPTER II.

VARIETIES OF ONSET.

Before attempting to consider the variations which take place in the stage of onset in typhoid fever, it is necessary to have some standard type of an average case of the disease in this period. The usual mode of onset, as described by Dreschfeld in Allbutt's *System of Medicine*, is as follows:

"In many ordinary cases the onset is insidious. The patient complains of pain in the limbs, of excessive fatigue, of cold and chilly sensations, of headache often very severe, of loss of appetite, and of sleeplessness. Epistaxis is a very common symptom, and generally occurs about the second or third day of the disease. These symptoms become more severe, the patient has to take to his bed, and from this day we generally reckon the duration of the fever. In many cases, however, as shown by the changes after death, the beginning of the morbid process must be dated from the very first symptom. The tongue becomes furred, and is at first moist; there is a steady rise of temperature, the evening temperature being generally one and a half degrees (F.) higher than the morning temperature, so that about the fourth day the temperature reaches 103° F. or 104° F.; the pulse rises to 90 or 100, rarely higher except in very severe cases, or in very young or debilitated subjects, is dicrotic and indicative of low blood-pressure; there is increased thirst; the abdomen is slightly distended and tender on pressure; diarrhœa may as yet be absent, and there may be constipation, or there may be two or three fluid stools from the first. Beyond headache, which persists for a few days, and sleeplessness, there are as yet no other symptoms; the skin is dry, but there are paroxysms of profuse perspiration. The spleen is as yet but little enlarged, and there are as yet no roseolar spots,

though when perspiration is profuse sudamina are noticed; the urine has febrile characters, and as yet does not show the diazo reaction. This stage lasts about seven days, and constitutes the first week of the enteric fever."

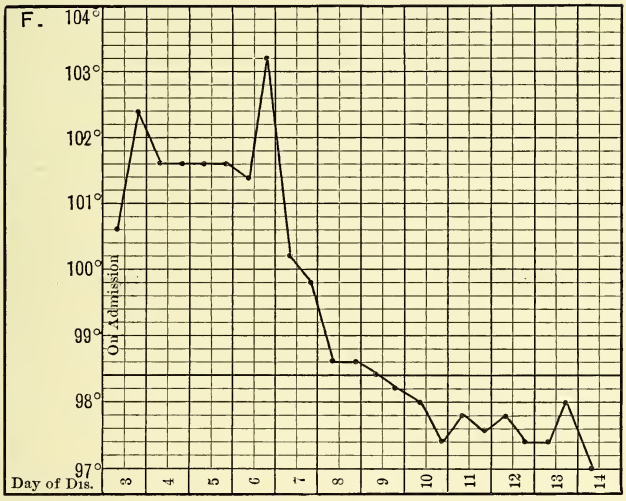
If this be taken as a type of an average case, we find at once that on either side of this type undoubted cases occur which by their extreme mildness may be overlooked, or by their great severity may mislead the physician into the diagnosis of some more acute and rapidly progressing affection. In the mildest of these cases there is little to be found indicative of enteric fever save, as Liebermeister puts it, "the long duration of an apparently trifling indisposition in which the patient presents a general impairment of health, malaise, physical and mental depression, and headache, with loss of appetite, the tongue being coated, and the pulse often distinctly slower than normal." No fever may be present. So moderate may all the symptoms be that a differential diagnosis between subacute gastro-intestinal catarrh and mild typhoid fever may be practically impossible except by the aid of Widal's test, which rarely gives results so early as the days of onset. Certain of the German writers have gone so far as to assert that all cases of subacute catarrh of this character depend for their existence upon mild typhoid infection.

Not only may the course of the malady be very mild indeed, but its length may be so brief as to throw doubt on its specific character, the whole illness lasting twelve to seventeen days, and then recovery being established. Sometimes even less time elapses before the fever ceases and the patient is manifestly convalescing.

Then, again, the abortive type of this fever presents itself, in which, after an illness beginning with quite characteristic manifestations, often of considerable severity, the symptoms rapidly ameliorate, and convalescence is established within ten days of the onset. This is well illustrated by the following temperature chart of a student recently under my care. On March 8th he first began to suffer from symptoms which were severe enough to make him seek medical aid and go to bed. Prior to this date he had felt but slightly unwell and this only for a few days. As is seen

in the chart, his temperature fell by crisis on the seventh day of his illness, although the positive Widal reaction endorsed the diagnosis of true typhoid fever. Curiously enough, such cases are often ushered in suddenly by marked signs—high fever and indications of grave illness—and yet so speedily pass on to the fall by lysis that it seems as if the attack must be due to some other infection. Such cases are recorded in which an initial fever of 106° in the axilla has been followed by a normal temperature as early as the seventh day.

Fig. 7.



Abortive typhoid fever ending by the seventh day, and by crisis instead of lysis.

In the malignant forms of infection the symptoms of onset may be of three types, viz., mild, followed by symptoms of increasing severity ; severe, with rapidly fatal developments, and, finally, aberrant symptoms pointing rather to the cranial contents, thoracic organs, or other parts of the body than to the abdominal contents. These various types will be found fully discussed in the following pages, but as an illustration of the cerebral type, a case reported by Green[1] may be cited. A child aged four years, had

[1] Australian Medical Gazette for August 29, 1897.

been quite well until four days before admission, when he was seized with an attack of giddiness while playing, turned around and around, and fell; but there was no loss of consciousness and no convulsive movements. Two hours later he vomited. There was no ear trouble. A week later, the seventh day of the attack, the child had a convulsion, lasting two minutes, which affected both sides of the body, and again on the eleventh day of his illness he had a very severe convulsion, lasting two hours, affecting both sides, although after it passed off there was marked twitching of the right side and conjugate deviation of the eyes to the right. The next day hemiplegia affecting the right side was well developed. The convulsions proceeded off and on for two days, affecting only the right side. Afterward vomiting became a constant symptom, and death occurred on the thirtieth day of illness and nineteen days after the first severe convulsion. At the autopsy a large portion of the temporo-sphenoidal lobe of the left side was discovered to be quite soft and pulpy, and on making a transverse section of this area the softening was found to affect the lenticular nucleus and to abut very closely to the anterior horn of the internal capsule. There was no hemorrhage, but the left middle cerebral artery was filled with a blood-clot.

When it is possible for a disease to present such widely various symptoms as have just been detailed, in its early stages, and when we are told by Liebermeister that "there is not a single symptom belonging to typhoid fever that is pathognomonic," it is evident that errors in diagnosis must occur even in the most skilful hands.

Temperature Variations from the Usual in Onset. Leaving the general consideration of the types of onset for a discussion of the individual symptoms of this period, we may take up the question of the range of temperature. The normal variation or character of the fever of onset has already been described in the preceding pages, but marked variations from that course are often present.

In this connection Dreschfeld quotes with approval a statement of Wunderlich's, which seems to the writer entirely too dog-

matic, in regard to the character of the oncoming fever, and it is certainly entirely at variance with more recent observations. I quote it to illustrate the older view of the disease: "Any fever which on the second day reaches to 104° F. is not enteric fever, nor is it enteric if the fever does not approach 104° F. on the evening of the fourth day; on the other hand, enteric fever may be diagnosed if in a middle-aged person suffering from an acute febrile attack the evening temperature on the fifth day, or within the first week, is between 103° and 105°, and alternates with morning temperatures, which are 1.4° to 1.7° lower, unless some other disorder can be discovered to explain the height of the fever. It is well to state that by morning temperature we mean the temperature about 9 A.M.; by evening temperature that about 6 P.M." These views certainly do not hold true to-day for the ordinary types of the disease. Attention has already been called to the very low temperature seen in the mild forms of the disease and to the high fever sometimes met with even in the so-called abortive cases.

During the stage of onset variations in the temperature of the patient may be due to complicating states which are about to be described, or they are perversions of the ordinary temperature of the initial days, occurring without assignable cause. The presence of a consolidation in the lung, of a pleurisy, or of a serious lesion in any one of the organs of the body, may entirely alter the chart in this period of the malady; and predominant localized symptoms may still further mask the case.

This is well shown by the following case recorded by Morris.[1] Aside from its obscure mode of onset this case is also of interest since, as a rule, hepatic infection manifests itself after an attack of typhoid fever rather than before:

On September 21, 1898, he was called in consultation by Dr. R. E. Doran, of Willard State Hospital, to see Mr. J. L. B., twenty-six years of age, who had been suddenly seized forty-eight hours previously, with a sharp pain below the right inferior costal margins, which rapidly extended as an acute general peritonitis, with

[1] New York Medical Journal, January 28, 1899.

a temperature reaching 102° F., but apparently without accompanying rigors. The patient was constipated until the day on which Dr. Morris arrived. On examination a mass was easily palpated at the site of the gall-bladder, and the peritonitis seemed to be most intense at that point. They diagnosticated empyema of the gall-bladder and operated. The peritoneum was deeply congested and was covered with coagulated lymph in the vicinity of the gall-bladder. The gall-bladder was distended with a mixture of thin, greenish mucus and thick, tenacious yellow pus. Dr. Morris did not have his culture-tubes at hand, and no bacteriological examination of the pus was obtained, much to his regret. He drained the wound and the gall-bladder with a small wick drain and closed the incision, excepting for the drainage opening. On the evening of the day of operation the temperature rose to 103° F. and dropped on the following morning to 100° F.; the pulse to 88; the respirations to 24. On the evening of the second day after operation the temperature rose to 106° F. Up to this time the bowels had not moved, but two high enemata of Epsom salts caused a number of loose movements, and the symptoms of dangerously progressive infection subsided rapidly. After this the symptoms of typhoid fever supervened, and the case ran a typical course as one of typhoid fever, ending in recovery in about four weeks, excepting for a small biliary fistula, which was closing spontaneously at last reports from Dr. Doran.

In nervous children or women the irritation of the heat centres often results in a sudden rise like that which is met with in the more acute maladies of an infectious type. And it is a well-known fact that typhoid fever in children is more apt to be ushered in by a chill and high fever than it is in adults, as has been well pointed out by Jacobi and J. Lewis Smith. A case of this character is reported by Guinon,[1] in which a child of two and one-half years was seized with high fever and with all the symptoms of pernicious malarial infection. Nine days later it suffered from collapse with all its characteristic symptoms, and the day following passed stools

[1] Revue Mensuelle des Maladies l'Enfance, 1897, p. 236.

which were typhoid in appearance. Collapse again occurred, and on the twelfth day symptoms of meningitis developed. Finally, a rose rash appeared, the spleen and liver were found to be enlarged, and the case proved itself to be one of unmistakable typhoid fever. The early age of the child, the sudden onset, the flushed face, the high fever, the collapse, and, finally, the meningeal symptoms are of interest.

In some instances in which high temperature is noted when the physician first sees the patient, it is not in reality the earliest perversion of normal temperature in that a mild and unnoticed fever has been present for some days, even though the patient has felt perfectly well.

High initial temperatures should place the physician on his guard, because they may mean severe infection or some grave complication which he must search for and discover, and particularly is this the case if the initial temperature is ushered in or is followed by a chill or rigor. In some of these cases careful study of the history of the patient will reveal an exposure to malarial infection, and an examination of the blood may reveal the presence of the malarial parasite, although, as pointed out further on, this organism is apt to be absent from the blood during the active period of typhoid fever.

The more sudden the appearance of the disease, and the more rapid the rise of temperature in the beginning of the first week, so much the more should one expect in general a short and even abortive attack, and the more rapidly the temperature falls, as the end of the first week is approached, the better the prognosis, particularly if the daily fluctuations are marked.

Very sudden development of true hyperpyrexia at this stage, unless it is due to some severe complication, is very rare.

Chills. In some instances, not commonly met with, typhoid fever is ushered in by severe chills. As already pointed out, these are most apt to appear in children, and they may indicate the development of some coincident infection. Chills may, however, be due to the typhoid infection itself. They are met with more frequently at the onset of a relapse than at the primary

onset. In a case recently under the writer's care, a man of thirty-five years, after several days of malaise, without fever, was seized with a violent rigor and at once became so ill that he was forced to go to bed, where he passed through a severe attack of the disease.

Under the name of "sudoral typhoid fever," Jaccoud records, in *La Semaine Médicale* for March 12, 1897, his belief in this special type, in which chills and sweats are prominent symptoms. The onset of the malady is sudden, and is accompanied by severe headache in the retro-orbital and occipital regions with shivering, fever, and sweats, so that the patient resembles one suffering from an intermittent malarial attack. These attacks are often quotidian and the febrile movement is hyperpyretic. The peculiar symptoms cease by the fifth day, and are followed by the usual course of typhoid fever. Quinine does no good in these cases, and they are not due to malarial infection. A second form is characterized by the primary appearance of headache and fever followed by sweating, which is profuse and asserts itself much later than in the form just described. The febrile movement is distinctly intermittent in type, but not so markedly so as in the form just named. In other cases, in place of a marked rigor, the patient has a subjective sensation of coldness in some part of the body, which can also be perceived by the physician if he touches the spot. In these forms the irregular manifestations may last three weeks and then gradually cease in the fourth week. Sometimes these cases are, however, very prolonged, and Borelli has reported instances lasting seventy or ninety days. Indeed, Jaccoud regards the length of the attack as characteristic. There are practically no complications. Albuminuria is extremely rare, but intestinal hemorrhage of mild degree is not uncommon. Peritonitis from perforation, Jaccoud asserts, is quite unknown in these forms, and he regards "sudoral typhoid fever" as a mild type of the disease. Notwithstanding the close resemblance of these types to double infection by the malarial organism and the typhoid bacillus, both Jaccoud and Borelli believe them to be pure typhoid fever, because they occur in persons who have never been exposed to malarial infection, and because quinine is useless.

The differential diagnosis is necessarily difficult in the early stages of the disease, although in general Jaccoud would have us believe that it is easy. It must depend largely upon the absence of any history of malarial exposure, upon complete development of most of the characteristic signs of typhoid fever, and, finally, upon the absence of any signs of the malarial organism in the blood and the presence of the Widal reaction. In cases of "abortive sudoral typhoid fever," in which the disease runs a very short course and stops abruptly, the diagnosis is very difficult. Jaccoud describes such a case as follows:

"In the patient referred to the headache and the temperature chart justified the diagnosis of mild typhoid fever, but the digestive organs were intact; there was no abdominal tympanism and no diarrhœa. The spleen was of perfectly normal size, the tongue a little dry, but otherwise showed absolutely none of the characteristics of typhoid fever. There was absolutely nothing in the lungs. The fever alone, and the slightly stupefied appearance of the patient, led us to assume the existence of some typhoid infection. There also existed on his body a measly eruption; but this was a superadded element, due probably to the large doses of antipyrine which he had taken, and also to some alcoholic frictions which had been given. Beside he was a grocer by trade, and grocers are specially exposed to skin irritations which not infrequently give rise to cutaneous affections. On the first days he had presented a certain degree of ocular catarrh, with redness of the conjunctiva and watery eyes. Then abundant perspiration appeared on the forehead, the nose, and the chest, drenching those parts completely. The fever developed in this way for ten days, the headache was general and persistent, but not very intense, and during the whole of this time there was nothing worthy of note, except the hypersudation and the rubeolar eruption.

"The case was evidently one of abortive typhoid fever of the sudoral variety, and could be classed in the mixed form which I have described. There was one abnormal point, viz., the subsidence of the fever, which was complete on the tenth day. Such rapid termination, not very unusual in ordinary typhoid fever, is,

I repeat, almost exceptional in sudoral typhoid. The differential diagnosis between sudoral typhoid and malaria—*i. e.*, typho-malaria, is, on the whole, easy, and hesitation between the two cannot last long, the administration of quinine salts, which are without action on sudoral typhoid, settles the question."

The violent headache of so-called sudoral typhoid fever, which is sometimes the only prodrome, may lead one to think of influenza, and in particular of the nervous form of that disease; but in influenza the pain is not localized in the head alone. It appears early and is very intense, but is also general all over the body; the temperature may remain normal, or, if there is fever, the temperature-curve is totally different from that of typhoid fever. The evolution of the influenza itself, which is in general of short duration when it remains uncomplicated, helps considerably in the differential diagnosis.

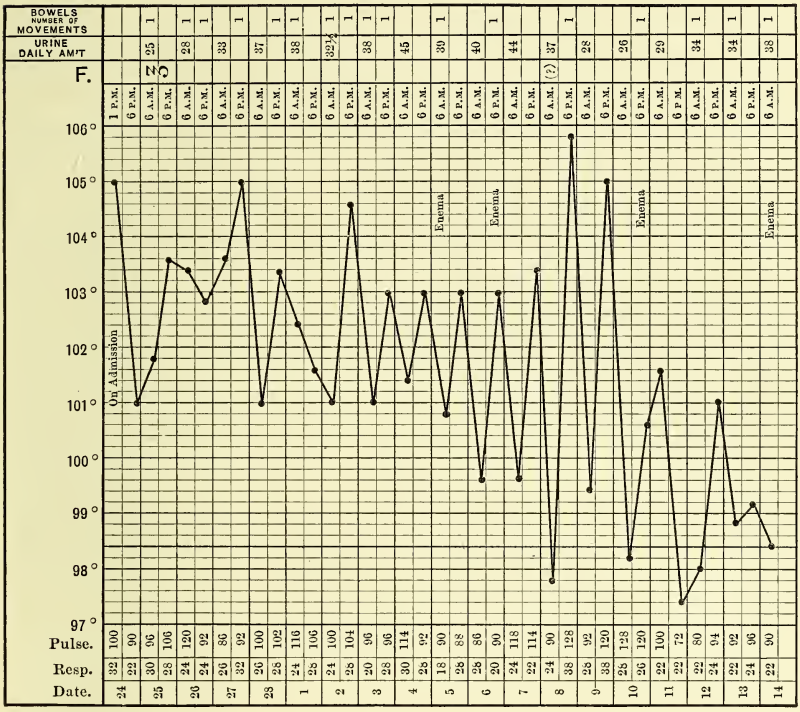
One might be misled into diagnosing measles when, along with the ocular catarrh, there is a discrete eruption of rose-colored spots, or else a true roseolar eruption like that of the patient under consideration. The absence, however, of all eruption on the face and neck, of broncho-pulmonary catarrh, the insignificance of the ocular catarrh, and the character of the temperature chart, all enable us, Jaccoud thinks, to eliminate this hypothesis without much difficulty.

The writer has had under his care during the winter, 1898–99, a case which followed this course:

A man of twenty-five years, a cigarmaker by occupation, was taken ill with what was supposed to be "malaria" or "grippe" on February 4th, but felt better and returned to work on the 6th. On the 7th he felt very ill, and entered my wards on the 8th. At this time he had marked swelling, as if from a phlebitis, of the left leg, which entirely disappeared in twenty-four hours. He presented all the characteristic symptoms of ordinary typhoid fever by the tenth day of the disease, but his temperature made the following extraordinary chart, each rise being followed by profuse sweating. He also had profuse night-sweats. He never had typhoid fever before, nor were there any signs of tuberculosis

or ulcerative endocarditis. His blood showed no signs of the malarial organism and gave the Widal reaction on the thirteenth day.

FIG. 8.



To save space this chart which showed in detail rigor after rigor and fever after fever, has been reduced to a morning and evening chart, and, therefore, only shows two or three paroxysms.

(For a discussion of so-called typho-malarial fever and of malaria complicating typhoid fever, see chapter on the fever in the well-developed stage of the disease, and that on other diseases which ape typhoid fever.)

Respiratory Conditions in Onset. Several cases have impressed upon me the fact that so-called "pneumo-typhoid fever" is a more common state than is generally thought, although it is true the standard text-books all describe this form of the disease. By pneumo-typhoid fever I refer to that form of typhoid fever in which the bacillus of Eberth exercises its primary influence

upon the pulmonary parenchyma, producing signs and symptoms which are practically identical with those of ordinary croupous pneumonia, even to the rusty sputum, although the usual rigor of onset, as seen in true croupous pneumonia may be absent or modified, and the onset in general is more insidious. In these cases toward the ninth or tenth day the high fever falls but slightly in place of the characteristic crisis, and when diarrhœa and rose spots appear, the possibility of the entire illness being due to a typhoid infection comes upon the mind of even the careful physician for the first time. This condition must not be confused with the so-called typhoid-pneumonia in which there is a double infection of the patient, his lung bearing the chief influence of the micrococcus lanceolatus and his intestinal canal and general system that of the bacillus of Eberth, nor the state in which the pulmonary consolidation results from asthenia or other causes incidental to the progress of an exhausting malady, and which is usually a catarrhal pneumonia or a congestion by stasis. As Osler has well said, "typhoid fever is a multiple infection in which the chief lesion of the disease may be found in other organs than the bowels," and, in a larger number of cases than is thought, pneumonia begins the attack of illness, and only later on does the character of the specific infection make itself manifest. The following case illustrates this fact very well, and is one of a number which have been met with by the author:

Z., a girl, aged ten years, was taken ill with a rigor and fever on November 10th, having been well enough to be up and out of doors at dancing-school the day before. The fever speedily rose to points ranging from 103° to 105°, and remained about these points for the first few days, when it gradually became a little less marked. It failed to respond readily to the use of cold spongings and the cold sheet, as a rule, although at times this treatment reduced it considerably. There was but little cough, and at times none of it for two or three days, but the child was somewhat dyspnœic, particularly at night, and cyanosis was marked. The pulse was usually as high as 120 to 130, and restlessness was constant. At times, particularly at night, there was delirium. An examination

of her chest revealed at the right middle lobe the physical signs of consolidation—that is, bronchial breathing, dulness on percussion, and absence of vesicular sounds, with exaggerated breathing elsewhere. At the left apex similar signs were present, and it was evident that the child had pneumonia. The facial expression, the somewhat dry lips and tongue, and the color of the patient's skin, combined with the fact that pneumonia sometimes is due to infection by the bacillus of Eberth, made Dr. Kirkpatrick, the physician who courteously called me in consultation, and myself cautious as to the diagnosis and the prognosis of the case, and, equally important, careful as to our treatment. The parents were told of the condition of the lung and of our suspicion that something other than a pure pneumonic infection was present, and we waited for the day of ordinary crisis with anxiety. On the ninth the temperature fell somewhat and seemed to give promise of relief, but on the next day it maintained its course; the tongue was found to be more enteric in appearance, and the rose rash of typhoid fever appeared on the chest and belly. Further, careful palpation and percussion at this time showed a slightly enlarged spleen and liver, an alteration in those organs not previously found, and diarrhœa, or, rather, looseness of the bowels, supplanted a tendency to constipation.

Under our older ideas of these diseases it would have been thought that a primary croupous pneumonia had merged into a typhoid fever by a gradual process of developing asthenia, or, again, that a double infection with the micrococcus lanceolatus and the bacillus of Eberth had taken place, whereas, at the present time we know that while such a double infection is possible, a single typhoid fever infection may result in primary pulmonary symptoms.

Still another case is that of B., a man of sixty-five years, who was taken ill with general malaise and wretchedness on a certain Friday. Fever and chilly sensations developed, but he kept on his feet for two days, when he was so ill that he had to go to bed. When seen by me in consultation on the fourth day of his illness there was rapid respiration (42 per minute), a pulse-rate of 120,

some cyanosis, a feeble, painful cough, and consolidation of the entire lower lobe of the right side, with exaggerated breathing on the left side of the chest. His temperature rose from 102° to 103°, and the bowels were costive to a marked degree. The sputum was rusty. A diagnosis of croupous pneumonia was made, and not until the tenth day of his illness did a persistent diarrhœa of ochre-colored stools, with spots, appear. The spleen had been found enlarged from the first visit that I paid him.

The difficulty in diagnosing these cases lies in the distinctly local manifestations and the fact that in some patients the fever may be quite high, delirium of an active form may be marked, and every symptom pointing to intestinal typhoid lesions may be absent. The question naturally arises as to the frequency with which this form of enteric fever occurs, but statistics concerning it are difficult to collect, since in many instances the condition is never recognized, or is recognized very late, and is not by any means always reported.

There is danger in these cases of still another error in diagnosis, and care must be exercised that a diagnosis of "pneumo-typhoid" is not made, when in reality the condition is one of tuberculosis of the lung, for in some cases of this character the rapid onset of fever, rigor, quickened respiration, cough, and the development of physical signs of consolidation, coupled with the continuance of fever after the time for ordinary crisis, will show that the disease is not croupous pneumonia. As a matter of fact, the cases of acute tubercular pulmonary consolidation simulating pneumonia at first or "pneumo-typhoid" afterward, are much more frequent than is pneumo-typhoid itself, and careful study of the case itself, or its history, and the microscopical examination of the sputum may reveal the tubercular character of the process. In all cases of suspected pulmonary tuberculosis, however, the absence of bacilli from the sputum will not negative the diagnosis of this malady, for until some tissue breakdown occurs the bacilli may not appear in the sputum.

It has already been pointed out that there is a form of pneumonia ushering in typhoid fever quite different in cause from that just

named, namely, that due to double infection with the specific organism of croupous pneumonia and that of typhoid fever. Such cases have been described particularly by Chantemesse. In such instances the febrile movement of the pneumonia merges into that of enteric fever. The early differential diagnosis of these two conditions is practically impossible unless, perchance, the bacillus of Eberth is found in the feces, which is not possible before the ninth day, or the Widal test gives a positive reaction, which it rarely does in the early days of the malady.

Acute pleurisy, like acute pneumonia, may usher in enteric fever, this condition being due to the ordinary causes of pleurisy being present simultaneously with typhoid-fever infection, or because of specific infection of the pleura by the typhoid bacillus. Thus Talamon[1] has recorded a case of enteric fever in which the onset was characterized by acute pleurisy, but the condition differed from that ordinarily seen in this affection by reason of the intensity and persistency of the fever, and by the general depression and sleeplessness, headache, and vertigo.

Talamon insists that there is a distinct difference to be noted between pleural-typhoid and acute febrile pleurisy, for in the typhoidal infection the symptoms are out of all proportion to the physical signs. The only condition which may closely resemble pleuro-typhoid is tuberculous pleurisy, but in tuberculous pleurisy the temperature is remittent, whereas that of typhoid is rarely so. Finally, the development of the other symptoms of typhoid will clear up the diagnosis.

A very much more rare respiratory disorder which may usher in typhoid fever is that chain of symptoms known as laryngo-typhoid, in which great hoarseness or aphonia develops with distinct evidence of acute laryngitis. These cases are quite different from those of severe ulcerating laryngitis seen in advanced stages of the disease, and which will be considered later on in the chapters on the well-developed and convalescing stages of the disease. Such instances are well illustrated by a patient described

[1] La Médicine Moderne, May 28, 1892.

by Bayer.[1] A physician presented himself for treatment because of aphonia and difficulty in swallowing, which was found to be due to acute laryngo-pharyngitis. These local symptoms were improved by treatment, but in a few days the man was seized with a severe chill, followed by fever and pain in the throat, an examination of which revealed a number of small superficial ulcers on the soft palate and on the pharynx; later the characteristic rose spots appeared on the skin. More interesting than all, particles of tissue removed from the heads of the ulcers just named contained the bacillus of Eberth. The inflammation extended to the ears, and deafness resulted. The patient finally died from intestinal hemorrhage and pneumonia. The finding of the bacillus in such cases would enable an early diagnosis to be made.

Almost equally rarely does a severe bronchitis usher in typhoid fever as a true pulmonary or primary manifestation, although, as the disease progresses, more or less bronchial inflammation is usually found.

Symptoms of Onset in the Kidneys. In very rare instances typhoid fever develops with marked evidences of acute nephritis, the urine being smoky or bloody in appearance, and containing albumin and casts. This form is sometimes called "nephro-typhoid," and by the French "*fièvre typhoide à forme rénale.*"

Gaillard[2] recently reported to the Société Médicale des Hôpitaux, for Bagot, the following interesting case of hæmaturia ushering in typhoid fever. The patient was a lad of ten and one-half years, who was taken ill on June 28th with hæmaturia. On July 3d the patient suffered from a good deal of tenesmus, pain in the urethra, and the urine contained red blood-cells but no casts. On July 7th distinct febrile movement was noted, the child complained of severe lumbar pains, which also extended into the limbs. He then passed through a typical attack of typhoid fever, reaching a normal temperature on July 26th, nearly a month after the onset of his attack. The urine contained no blood after the eighteenth day of his illness. Bagot asserts that there is no doubt

[1] Revue de Laryngologie, d'Otologie et de Rhinologie, July 15, 1893.

[2] La Presse Médicale, February 11, 1899.

whatever about the correctness of the diagnosis. That this patient had a distinct tendency to hæmaturia seems indicated, however, by the fact that in subsequent illnesses, other than that due to the typhoid infection, he also suffered from this condition of hæmaturia. (For further remarks see later chapters.)

Retention of urine is sometimes met with in the early stages of typhoid fever but usually passes away in a few days.

Symptoms of Onset in the Alimentary Tract. Tonsillar inflammation, associated with severe pharyngitis, sometimes begins the course of enteric fever, and escapes correct diagnosis as to its cause for a considerable period of time because of the situation of the lesions, and also because tonsillitis of an active form is so commonly associated with marked evidences of general systemic infection, the patient oftentimes appearing profoundly ill and suffering from general wretchedness, febrile movement, a heavily coated tongue, impaired hearing, and mental hebetude.

A case of this character is under my care in private practice at the present time. A woman of thirty years was taken ill with what appeared to be a severe attack of acute tonsillitis with high fever. As the fever failed to disappear with the subsidence of the tonsillar swelling and pain, and as an epidemic of typhoid fever was present, her blood was examined for the Widal reaction, and it was found, and simultaneously other symptoms of enteric fever developed.

A peculiar form of ulceration of the pharynx has been recorded by Bouveret,[1] Devignac, Dengnet, Wagner, and Cahn. They call it "pharyngo-typhoid." The ulcers are superficial, clean-cut, and appear chiefly on the soft palate. (See also later chapters.)

(For œsophageal lesions see the next chapter.)

Probably the most common perversions of the early manifestations of enteric fever are to be found in association with the functions of the gastro-intestinal tract. So common are they, and so localized are the dominant symptoms in these cases, that the malady seems quite distinct from true typhoid fever, and is often

[1] Berliner klin. Wochenschrift, 1885, No. 14.

called the gastric form of typhoid fever. In some instances, it is true, fever of mild degree develops in cases of gastric catarrh of a more or less severe form, but they are not characterized by the profound degree of illness seen in the gastric type of enteric fever, in which persistent vomiting and epigastric disturbance followed by diarrhœa are the main symptoms in the early or initial stages. Such gastric types are more commonly met with in children. As well pointed out by Bristowe, undoubted enteric fever in childhood, at which age recovery commonly occurs even if the disease is overlooked, is often called, for want of a better name and a certain diagnosis, by the conscience-quieting term of "infantile remittent fever," "bilious fever," and "gastric fever," or even "worm fever." (See Frequency of Enteric Fever in Childhood, in Chapter I.)

The gastric manifestations when severe are, perhaps, more rapidly discovered to be due to enteric fever than if the infection be mild when the other typhoid symptoms are not marked. These gastric symptoms are rarely met with in the great cities of the eastern part of the United States, and vary in different epidemics, although they are asserted by Murchison to have been commonly met with in his experience. On the other hand, Hutchinson, in his classic article in Pepper's *System of Medicine*, tells us that these acute gastric symptoms with nausea and active vomiting have been unusual in his experience. When vomiting ushers in the disease in a child it does not seem to be as evil a prognostic sign as when this symptom begins the attack in an adult. I saw a year ago a case, in consultation with Dr. Orville Horwitz, in which persistent vomiting was the first sign of the disease, and preceded a very severe illness. Vomiting in a child is readily produced by any disturbing ailment, but in an adult it probably results from a more or less profouud infection, and rapidly causes exhaustion if it is persistent, as it is apt to be in this class of patients. When the vomiting is mild, or, in other words, is repeated but once or twice, it is not, of course, of any gravity, and no less an authority than Murchison intimates that such cases often seem to be benefited by it if it be not too persistent.

A severe and continued vomiting attack in a case free from malaria and associated with persistent febrile movement ought to arouse the suspicion of typhoid infection to a sufficient degree to cause the physician to be on the watch for further confirmatory symptoms, particularly if the illness is not relieved by the ordinary measures utilized for the cure of such an illness.

Another variety of onset, represented by disturbance of the gastro-intestinal functions, is that characterized by the sudden development of violent diarrhœa of the serous type, instead of the constipation usually met with during the first week of the disease. Such cases are not common, but are represented by the following case in my own experience. A man of thirty-five years, apparently in perfect health, and whose appetite had been excellent up to and including the morning of the beginning of his illness, began to suffer after a moderately heavy luncheon from slight headache, which he attributed to indigestion, to which he was subject. He ate no supper because of nausea, and was seized at twelve o'clock midnight with an active, watery diarrhœa, resembling a mild attack of cholera morbus, in that the abdominal pain was not very severe. No vomiting occurred. By the use of chlorodyne in full doses he was able to remain out of bed for four days, but at the end of that time was seized with a severe rigor followed by moderate fever rising to 104°. He then developed mild typhoid symptoms, but, ten days after the fever ceased, suffered from a severe relapse. It was found that just thirteen days prior to the diarrhœa he had eaten raw clams contaminated by sewage, and that eight other persons who ate of the same lot of clams also had the disease. The active diarrhœa in this case, followed by wretchedness and general malaise, was naturally supposed to be in no way connected with a definite and specific infection.

Still another case of this kind is that of a patient admitted to my wards with a history that up to January 16th he had been in good health, but on that day, while working in a sugar-house, and exposed to high temperature, he had taken large draughts of cold water, which speedily produced symptoms of cholera morbus, followed by headache and anorexia, and these again by the early

symptoms of enteric fever, which caused him to come under my care a week later with, as additional symptoms, signs of congestion of the middle lobe of the right lung. Rose spots appeared on the ninth day of his illness.

Pepper and Stengel[1] have reported seven cases of abrupt onset in typhoid fever, and they assert that Moore, in his *Text-book of Eruptive and Continuous Fevers*, published in 1892, is the only authority who calls particular attention to these cases in which the disease begins abruptly and with vehemence, characterized by decided rigors, violent headache, and rapid rise of temperature. Moore thinks that the whole course of the disease is becoming more typhus-like than formerly. Pepper and Stengel's seven cases may be divided into two classes: those in which the preliminary symptoms were simply gastro-intestinal in character, vomiting, purgation, and high fever being present, and others in which violent headache and catarrh of the throat, nose, and bronchial tubes was marked.

Symptoms of Onset Connected with the Nervous System. Of the nervous manifestations of typhoid invasion three chief types may be mentioned, namely, (*a*) that in which the patient suffers from delusions or aberration of mind and wanders from home until he becomes so ill as to fall and be taken to a hospital, or, perhaps, loses his life through exhaustion, or accident due to his stupid mental state, or by means of deliberate suicide. (*b*) The second class is that in which acute maniacal symptoms ensue. (*c*) The third class in which evidences of meningitis are marked; so marked that true meningitis is supposed to be present, or in its place meningitis secondary to croupous pneumonia. In many of these cases there is little doubt that the pulmonary lesions of typhoid infection are responsible for the meningeal signs, while, on the other hand, it is possible for direct infection of the meninges by the typhoid organism to occur, although this is rare. (See further on.)

Some years ago myself and Patek collected the following cases

[1] Philadelphia Medical Journal, vol. i. No. 2.

of mental disturbance in onset which we[1] found in the literature of the subject:

Murchison[2] reports the case of a German who was much excited over the Franco-Prussian War. After about four days of discomfort and malaise, he suddenly passed into a state of acute maniacal delirium, requiring two men to control him. There was an absolute refusal of food, a temperature of 102°, with a dry tongue and rapid pulse, slight diarrhœa, and no spots. The patient was subdued by large doses of chloral, and the fever ran its course. The same author also states that in several instances he has known acute mania to develop on the first day of an enteric fever, and that under these circumstances the case is very apt to be mistaken for insanity.

Wilson[3] asserts that delirium may be an early symptom of enteric fever, and quotes Riberalba, who reported four cases which were delirious on admission to the hospital. Louis saw two cases which were delirious on the first night of their illness. Bristowe has also reported a case in which maniacal delirium existed on the second day. Mottet mentions an instance of typhoid fever complicated with mania to such a marked extent that the patient was placed in an asylum before the true nature of the ailment was discovered, and Henrot and Bucquoy have seen the disease ushered in with the delirium of grandeur. Finally, Daly[4] records an instance in which aggressive mania came on on the fifth day, following a condition of stupor.

From a careful examination of a large amount of literature I am convinced that prodromal insanity in enteric fever is most rare and, when it occurs, is almost always fatal, while the insanity which is in the nature of a sequela may be looked upon as devoid of immediate or remote danger to mind or body.

In very rare instances delirium may be almost the first symptom of typhoid fever. Indeed, it may actually precede the devel-

[1] Hare and Patek, Medical News, 1892.
[2] Lancet, 1870, vol. ii. p. 807.
[3] Philadelphia Medical Times, 1884–85, vol. xv. p. 577–581.
[4] The Medical News, 1882, vol. xl. p. 68.

opment of pyrexia; thus in seventeen cases which have been collected from literature by Aschaffenbourg,[1] seven were characterized by the development of delirium before the fever, and the latest period at which it was observed among these cases of early delirium was the end of the first week. As a rule, the delirium lasted only a few days, but the mortality was high, six of the seventeen patients dying. Among these cases the delirium occurred in two forms, either the patients were exceedingly restless and violent, finally becoming torpid, or there was a condition of confusional insanity, in which the patients sang, prayed, danced, or were gay or sad.

The following cases met with by Patek and myself are of interest: Annie M., aged twenty-four years, was admitted to St. Agnes' Hospital, March 18, 1891. She had been feeling badly for some time, but until four days previously had been able to do her work. On the 14th she had a severe headache, vomited a little, suffered from pain in the stomach, and had some diarrhœa, these symptoms being followed on the subsequent day by not very profuse epistaxis. She walked a considerable distance to the hospital, and on her admission, at 10 P.M., her temperature was found to be 105°. The resident physician found that her tongue was thickly coated, dry and brown. On the next day when seen by us in the wards the tongue was unusually clean even for that of a healthy person. The patient was delirious and so violent that it required four or five persons to keep her in bed. The temperature, after an unusually prolonged and severe struggle, was found to be 106°.

At this time every symptom of typhoid fever was completely masked by the insanity. The bowels were moved and the passages were of normal consistency and color. The urine was somewhat scanty and high colored, and the pulse full and strong. There were no rose spots or other enteric symptoms. At the end of twenty-four hours the patient, still being in a condition of wild insanity was removed to a cell, the impression being that it might be

[1] Archives de Neurologie, March, 1895.

a case of hysterical mania with hyperpyrexia. Twenty-four hours later the insanity had disappeared, and the typhoid symptoms asserted themselves; the delirium became more quiet and muttering, and she was taken back to the wards. During the following week she was constantly delirious, and frequently maniacal, although there were short momentary intervals of sanity. During this time a large number of rose spots appeared on the abdomen and chest, the tongue became heavily and typically furred, the temperature followed a characteristic course, the typhoid odor was present, and an occasional nose-bleed helped to confirm the diagnosis of typhoid fever. The patient rapidly became worse, and died thirteen days after admission, without becoming sane, except for the brief intervals named.

The second case is as follows:

Mr. A., a resident of Milwaukee, aged thirty-four years; married; one child. A sister died of convulsions of unknown nature but a short time before the onset of his illness. Family history otherwise negative. At the age of seventeen years the patient, according to the statement of his physician, had an attack of typhoid fever, attended with as much, if not more, delirious excitement than this, the second attack. The history of the case begins with the circumstance that Mr. A. was nursing his wife, who was down with a mild attack of typhoid. The patient's first complaint was of headache and insomnia. The visiting physician, seeing him on the following day, ordered him to bed, recognizing the case as one of typhoid fever, rather because of the existence of a like case in the same house and from the mere complaint of malaise, than from any symptoms particularly characteristic of the disease. The patient obeyed the instructions of the physician, and went to bed, still complaining of insomnia. Hardly had he fallen into a mild slumber when, not more than an hour later, he suddenly awoke, delirious, and grew steadily more so. During the following night he became maniacal, rushed to the room of the nurse (she had been procured since the husband's illness), burst open the door, threw the nurse to the floor, and assaulted her in a most violent manner, kicking and striking her, and accusing her

of wishing to harm his wife and child. The nurse finally managed to escape, and ran for the physician, who lived across the street. In the meantime the patient jumped through a window leading to a small balcony over the front portico, and leaped to the ground, where he was found a few minutes later by the physician. Strange to say, the man suffered little injury, being slightly bruised by the fall, and somewhat cut by the glass; but stranger still was the fact that he was now quite rational, telling the physician all that had transpired and what he had done. The patient was again put to bed, now apparently quite comfortable. The physician left him to see the wife in an adjoining room. Hardly, however, had he gone when Mr. A. suddenly sprang from the bed, rushed into the kitchen, where he seized a large knife, and then rushed back, bent upon assaulting the physician. He was, however, overpowered and again forced to bed. He now rested comfortably, and when seen the following day was doing well. That evening a condition of hyperpyrexia suddenly intervened, and in a few hours the patient was dead.

The following case is of interest in this connection, and was seen by me through the courtesy of Dr. Higbee, of Philadelphia, who called me in consultation.

An unusually large, muscular man, about thirty-five years of age, after two or three days of wretchedness and malaise, with slight headache, developed fever of moderate degree on the fourth day, and that evening became maniacally delirious, so that it required four or five of his fellow-workmen to hold him in bed. On these workmen becoming exhausted the following night two male nurses were put in charge of him, but he fought them so vigorously that they refused to take care of the patient when the morning arrived, as they stated he was so powerful that he threw them all about the room.

When I saw him after two nights of violent delirium of this character, he was perfectly himself, mentally, and described his condition and his sensations to me, using unusually good English for a man in his walk of life, and evidently having an intelligent idea of the chief symptoms to which he was subject. He had no

recollection of his delirium, but he had been told by his wife of the struggles that they had had with him on the previous night.

An exceedingly careful examination of his chest revealed at the apex of the right lung, anteriorly, a small patch where there was impaired resonance and the other physical signs of pulmonary consolidation, and after consultation, Dr. Higbee and I agreed that it was one of those cases of pneumonia in which there was a remarkably small pulmonary lesion, accompanied by severe meningeal and cerebral symptoms. Something about the case, however, made me suspicious of a typhoid infection, and I stated to Dr. Higbee that while there were no symptoms of typhoid fever present that I could point to, I was suspicious of the development of this disease. That evening the man again became maniacally delirious to such an extent that his family recognized that it was impossible to keep him at home, and he was admitted to the hospital, where he died in forty-eight hours from exhaustion. The autopsy revealed typical typhoid ulceration of the bowel and other pathological evidences of well-marked typhoid fever.

This case illustrates very well not only the fact that pneumonia and typhoid infection may exist side by side, the pulmonary condition being, perhaps, directly due to the infection of the bacillus of Eberth, but also that cerebral symptoms of great severity may usher in both typhoid fever and pneumonia.

Osler records two cases of curious aberrant mental state in the stage of onset. In one, a young girl began her illness by doing odd things and having laughing and crying spells; the other, also a young woman, was distinctly "off her head," so that she was regarded as a pure mental case.

There is still another nervous type of onset which is exceedingly rare, namely, that of rapidly developing stupor and coma.

Very rarely in children the disease is ushered in by a convulsion, as in a case recorded by Osler, and in the case of convulsions reported by Green, and detailed in an earlier part of this essay. Convulsions when met with in adults are usually seen in the later portions of the disease, and depend upon embolism or thrombosis of important cerebral vessels.

The Skin in the Stage of Onset. As is well known, the characteristic rash of typhoid fever does not make its appearance, as a rule, until the seventh or ninth day, and, therefore, it cannot be considered a symptom of onset in typhoid fever. Cases do occur, however, in which in this stage of the disease aberrant rashes develop. Thus the writer has under his care at the present time a man of twenty-two years, who entered the hospital on the third day of his illness so covered by a profuse scarlatiniform rash that a differential diagnosis as to its true character was impossible. It persisted for three days, and then gradually faded, and the case ran a course of typical typhoid fever. (See the chapters on the skin in the well-developed and convalescent stages.)

CHAPTER III.

THE ABERRANT SYMPTOMS, STATES, OR COMPLICATIONS OF THE WELL-DEVELOPED STAGE OF THE DISEASE.

Temperature in the Developed Disease. We may pass on, then, to a consideration of excessive symptoms and complications of the developed disease, and the febrile process naturally first attracts attention. Before we attempt to study the unusual febrile conditions seen in patients who have passed the stage of onset and are in the well-developed period of the malady, it may be well to consider briefly what the normal or usual febrile movement really is. This Strümpel well describes when he says that the second division of the curve represents the so-called fastigium, and corresponds to the height of the disease. "During this time the fever presents in most of the severer cases the general character of *febris continua* —that is, the spontaneous remissions of the fever seldom exceed 2°. Almost always the lower temperatures come in the morning hours and the higher in the evening. In cases of average severity the morning remissions touch 102° to 103°, and the evening exacerbations 104° to 105°. Temperatures which reach or exceed 106° are seen only in very severe cases. Considerable morning remissions are always a favorable symptom, while morning temperatures of 104° or higher generally show the case to be severe. The duration of the fastigium varies with the severity and obstinacy of the case. It may last only a few days or one and a half to two weeks; in violent cases still longer."

Ampugnani[1] has proved that the natural maximum occurs between 3 and 6 P.M., and the natural minimum between 5 and 8 A.M.

At the end of the fastigium the temperature gradually falls

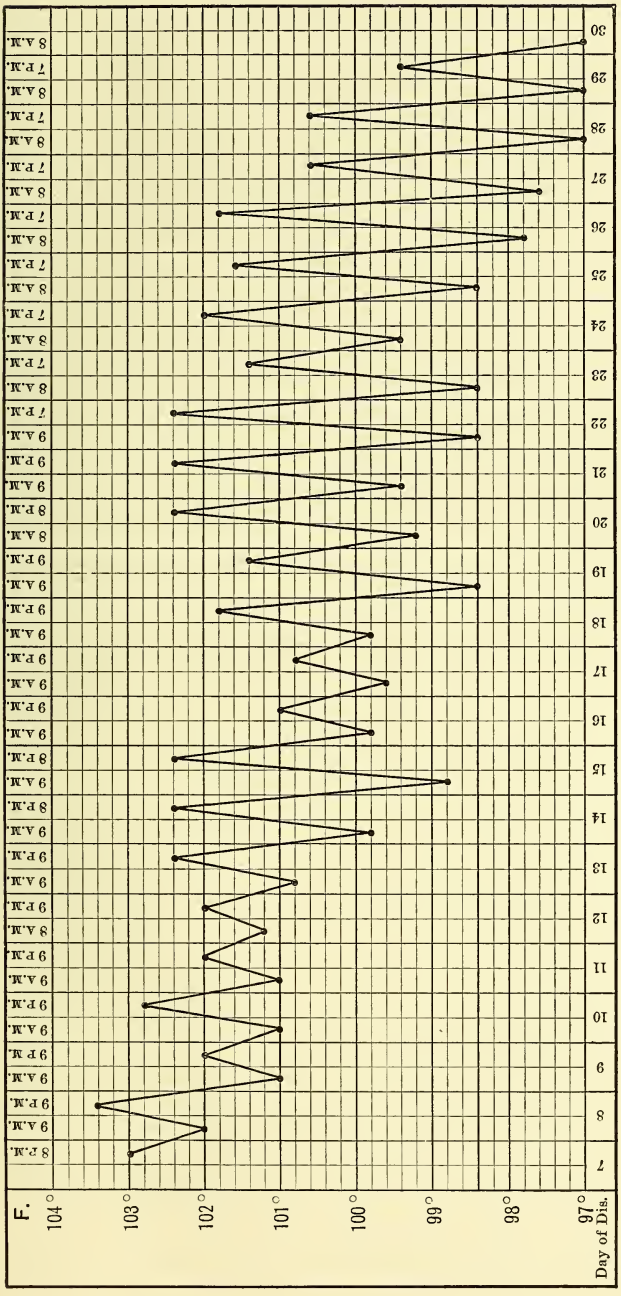
[1] London Medical Record, January, 1889.

by lysis until it reaches the normal, or perhaps more frequently there is before the lysis another period which has been called by Wunderlich the "ambiguous period," in which the morning temperatures are each day almost normal and the evening temperatures only slightly lower each day. In other cases the evening temperature for some days remains as high as before. Murchison called this period "the stage of changing fortunes," and Strümpel has called it "the period of steep curves," and has also stated that the longer a case lasts the more marked becomes the irregularity of the fever at this time.

The case recorded in this chart was one of very great interest, because as the fever of the early stage of the disease was not marked, and the abdominal symptoms were prominent, the question arose as to whether the patient, who was five months pregnant, was suffering from appendicitis, uræmia, sepsis from pelvic disease, septic endocarditis, or typhoid fever. There was scantiness of the urine, half the normal amount of urea, albuminuria, and marked signs of general toxæmia. There was also great tenderness of the belly, particularly over the appendix, and considerable pain in this region, even when the patient was lying still. In addition there was also great difficulty in urination and obstinate constipation, and the pregnant uterus so filled the lower segment of the belly and displaced the bowels that diagnosis was unusually difficult. Auscultation over the præcordium revealed a distinct endocardial murmur, probably due to the anæmia of pregnancy. Had these steep curves been met when the patient was first seen I think the case would have been considered one requiring operation, because they would have led me and the surgical consultant to believe that the symptoms were septic. The development of a profuse rose rash and the Widal reaction cleared the diagnosis some days before the period of steep curves began.

Having set up a normal standard for the course of typhoid fever, we find that variations from this standard occur under circumstances, many of which are indicative of some condition well worthy of the physician's attention, while, on the other hand, some aberrant types are without significance so far as our present knowledge

FIG. 9.


F.
104°
103°
102°
101°
100°
99°
98°
97°
Day of Dis.
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30
8 A.M. 1 P.M. 8 A.M. 1 P.M. 8 A.M. 1 P.M.

goes. The temperature of typhoid fever is, as is well known, rarely as high as in many other of the grave infectious maladies, yet at times it may become in itself dangerous by reason of its height. Sometimes, though rarely, as in the days of onset, we meet during the fastigium, without the presence of an additional exciting cause over and above the ordinary typhoid infection, with cases in which there is developed a distinct hyperpyrexia amounting to 105°, or even, very rarely, to 110°.

Such high temperatures are sometimes seen for long periods of the attack as the result of nervous excitement, or of unusual susceptibility to the infection in the sense that the heat mechanism is easily disturbed by the disease. These cases, as a rule, however, do not persist in hyperpyrexia, but soon fall to the usual level. When the fever is persistently high there can be no doubt that, as a rule, the attack is one of a severe character. Conversely, a low range of fever is indicative of a mild attack, although by no means proof of it, for moderate fever is sometimes seen in cases characterized by very severe infection. Rarely the disease, pursuing a fatal course, is accompanied by progressively rising fever until toward the end of the second or third week it may reach 107° or even 110°, as has been recorded by Wunderlich.

When a severe and prolonged attack of typhoid fever is present the period of "steep curves" may be postponed from the end of the third or beginning of the fourth week, or even to the fifth or sixth week, and in these cases there is usually wide-spread ulceration of the small and large intestine. Additional evidence of this condition is adduced by the fact that the abdomen is still tender on pressure, and the so-called meteorism or active peristaltic movement is persistent. Care must be taken in these cases that other causes than uncomplicated typhoid fever are not actively engaged in the continuance of the fever, either in the form of other infections or as secondary infections by the bacillus of Eberth of such parts, for example, as the gall-bladder, the kidney, or the bones. Or, again, the fever may be continuous as the result of a tuberculous infection superimposed on the typhoid trouble or antedating that disease in time of entrance into the body, but only active

when vital resistance is decreased by the exhaustion of typhoid fever. (See further on.)

Among the particularly noteworthy causes of sudden rises of fever during the fastigium, or in the period of ambiguity, or during lysis, we find the development of some acute complication, such as pneumonia, catarrhal or croupous, abscess in some part of the body, and what has been called "intercurrent relapse." The pneumonia at this period is often of the croupous type (8 per cent.), and pleurisy may also develop (8 per cent.), but their onset may not noticeably disturb the temperature curves, so that while the presence of a rise may be indicative of another source of difficulty, its absence does not indicate that no secondary pulmonary trouble has arisen; more rarely still catarrhal pneumonia elevates the temperature, and its very insidious onset makes it readily overlooked, while the development of hypostatic congestion may make no change at all. The temperature under some circumstances rises quite suddenly, and, after maintaining a generally higher course for a few days, begins to drop back to its former level, or at once the whole temperature course passes into the stage of lysis. So, too, an otic abscess may produce such results, and, finally, should an intercurrent relapse ensue, the fever, gaining new force, may mount to a point as high or higher than any previously reached, and last from ten days to two weeks or more, falling again as a tendency to lysis is developed. The presence of a mild primary attack followed by a relapse after several days of no fever, and finally complicated by phlebitis, with fever secondary to it, and then a second relapse, is shown in this chart.

It is important that a secondary exacerbation of the fever be not regarded as indicative of true relapse unless it persists, unless it is followed by a renewal of many or all of the earlier symptoms of the disease, and unless the eruption and enlargement of the spleen a second time indicate true secondary infection. Not only is the physician to avoid a diagnosis of relapse until it is proved to be present, for the sake of accuracy, but in addition he must avoid it, because it is an easy way to explain temperature irregularities, which should cause him to care-

FIG. 10.

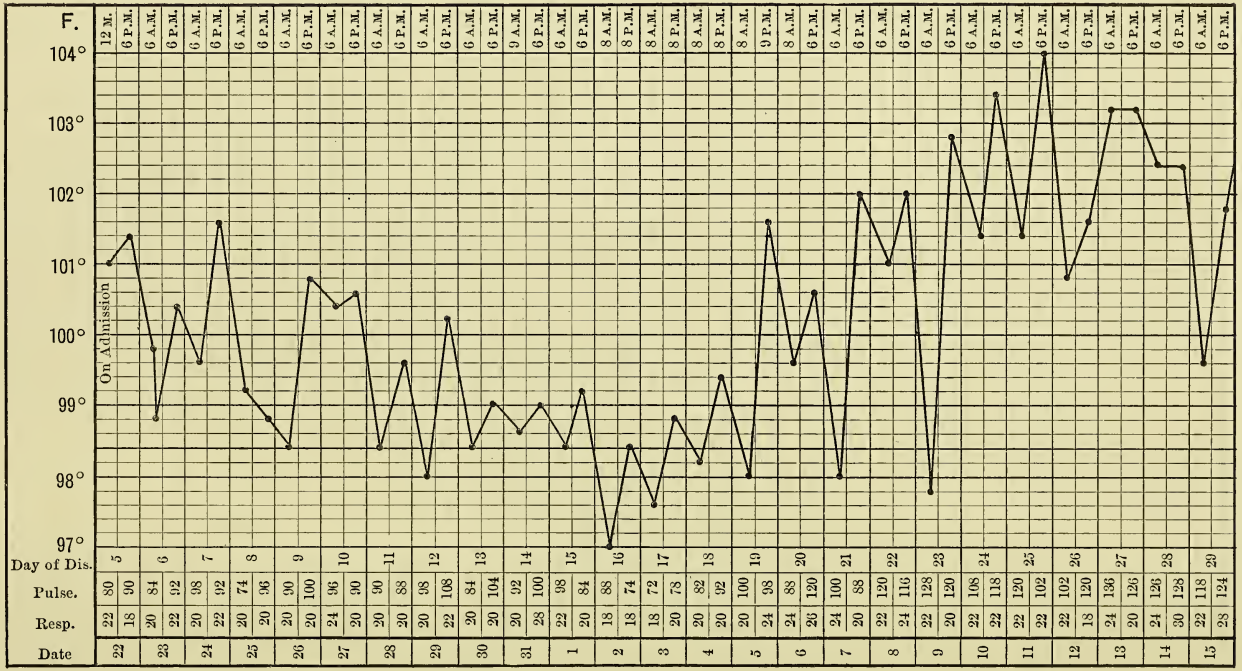


Chart showing mild typhoid fever for twelve days, followed by a relapse lasting nineteen days, this in turn by phlebitis of the left leg, causing a sharp rise on the thirty-eighth day, and this again by a second relapse which ends by crisis on the fiftieth day of illness.

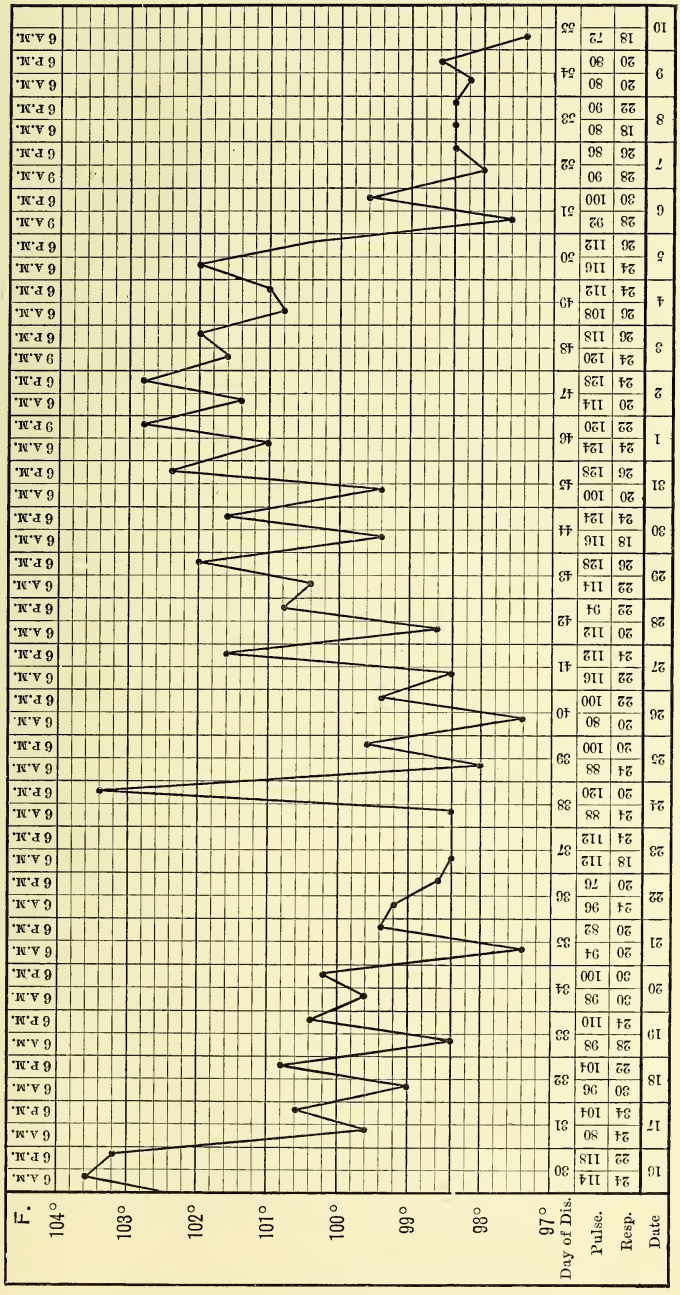


Fig. 10 Continued.

fully search for complicating affections. To sum up this matter with brevity, it should be the rule to consider any sudden and considerable rise of fever above the ordinary lines previously followed, an indication of some other factor than the ordinary typhoid infection. These various complicating states which are productive of febrile movement will be discussed later on when studying the lesions found in various organs.

Of the cases in which the temperature is of low degree and mild, much may be said. In the first place, in very rare instances cases occur in which there is not only no fever, but actually a condition of subnormal temperature from the beginning to the end of the attack. Thus in several cases under the writer's care, some years since, there was a characteristic temperature curve in form but not in degree, the morning temperature being distinctly subnormal and the evening temperature normal, and in which the return to health consisted in a "lysis," so to speak, in which the temperature gradually rose to normal instead of falling. Again, almost equally rarely there is no temperature movement whatever in the sense that the temperature is either above or below normal.

Cases of this type have been recognized for many years by close students of the disease, but are not commonly recognized by the general practitioner, who is taught in the medical schools to regard fever as a necessary symptom of this malady. Many years ago the elder Miescher recognized these cases, and Liebermeister recorded, in 1869, 139 cases of "afebrile abdominal catarrh," which he thinks were in large part due to typhoid infection, and, in 1870, 111 cases of the same character. Many of these cases showed evident enlargement of the spleen, and in some instances a roseola. Strabe[1] has described fourteen cases in which no fever was present, although at times the temperature was subnormal, and in which, nevertheless, the other characteristic symptoms of enteric fever were present to so marked a degree that they could not be mistaken for any other disease. The mortality in these cases was

[1] Berliner klin. Wochenschrift, 1871, No. 30.

no less than 14.1 per cent. So, too, Fraentzel[1] has recorded forty-one cases treated in a field-hospital during the Franco-Prussian war, in three of which the fever did not exceed 99.1°, and in the rest did not arise above 102.2°, and yet in which the mortality was 39 per cent. for the forty-one patients. Guitéras[2] records a case in which he diagnosed the condition as intestinal obstruction, in which the patient died of peritonitis, and at the autopsy the lesions of typhoid fever was found, although no fever had been present. Vallin[3] records a case of death due to perforation in an afebrile typhoid fever patient, and another of intestinal hemorrhage in a similar case, and the writer has seen several afebrile cases in one epidemic. In still another epidemic another instance was met with, which has been recorded in the Memphis *Lancet* for July, 1898. (See further on.)

In *La Province Médicale*, November 26, 1897, Weill and Piery report a case of apyretic typhoid fever, which they considered in other ways entirely typical.

Two cases of apyretic typhoid fever have also been recorded by Wendland.[4] These cases were confirmed by autopsy, and illustrate, at least to the satisfaction of Wendland, that temperature is not a true index of the severity of the disease.

Similar cases have been recorded by Fisk, of Denver, and they are represented by the following case:

The patient was a male with a negative history, except that he had true typhus fever at ten years. On admission he had a temperature of 98.4°; pulse, 84; respirations, 26; the tongue was coated, showing distinct red tip and edge; he had an apathetic appearance, and complained of headache; the pupils were dilated, there were tenderness and gurgling in the right iliac fossa. He still had constipation, but when by medication the bowels were acted upon, the fecal matter was of pea-soup color and liquid.

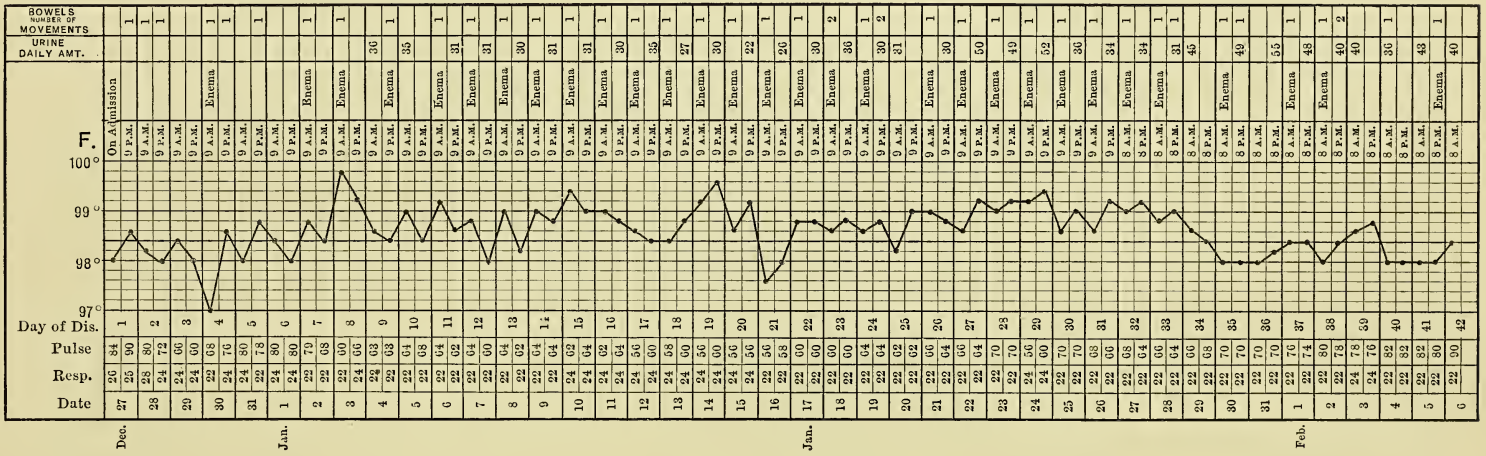
[1] Zeitschrift für klinische Medizin, 1881, p. 226.

[2] Transactions of the Association of American Physicians, 1887.

[3] Archives Générale de Med., November, 1873; see also Liebermeister and Hagenbach Aus der med. klin. zu Basel, 1869, p. 9.

[4] Deutsche Medizinal Zeitung, August 29, 1893.

FIG. 11.



Temperature chart from a case of afebrile typhoid fever.

There was an eruption of "rose spots;" the spleen was normal. Upon the patient's abdomen and back were found numerous pale-blue spots—*tache bleuâtre.* Close inspection also showed evidences of pediculosis, several ova being attached to hairs.

Later it was noted that the spleen was slightly enlarged, also that the palms showed the characteristic yellow tint; constipation still existed, but the pulse was not so rapid as on admission.

The urine was yellow; specific gravity, 1020; acid, no sugar, no albumin.

Later the headache nearly disappeared, but stupor still continued. The diagnosis was afebrile typhoid.

The accompanying temperature-chart is an interesting confirmation of this history:

Dreschfeld also mentions this form of apyrexial typhoid fever.

Under the name of *typhus levissimus,* Griesinger first described forms of enteric fever in which the febrile movement was not only very mild, but in which the symptoms in general were of the most moderate form, the entire course of the disease lasting only eight to fourteen days.

In that condition known as "abortive typhoid fever," the severe onset and high fever may so soon be followed by moderations and signs of convalescence, with a falling temperature, that the course of the temperature may be most aberrant and the chart misleading.

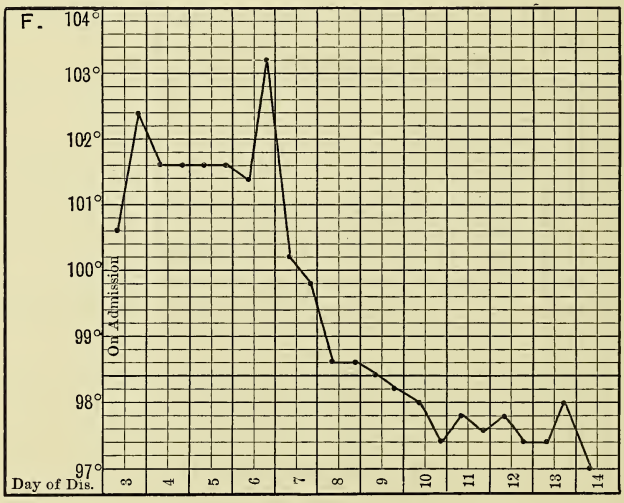
Here, again, however, as in all the variations of temperature just described, the physician must not be readily led into a diagnosis of an aberrant form of typhoid fever by the knowledge that such aberrant forms occur, for these forms are so infrequent as to be curiosities, and are so rare that the probabilities in an obscure case are against their presence. Only the clear and undoubted development of a sufficient number of pathognomonic symptoms coupled, if possible, with a positive reaction with the Widal test and with a history of recent possible typhoid infection should cause the physician to reach a diagnosis of these types of enteric fever.

In aged persons enteric fever is usually mild in its temperature

curves, and the characteristic febrile movement is so irregular and distorted as to be devoid of much diagnostic value.

In some cases the fever is peculiar in that it fails to follow the so-called normal rise in the evening and slightly lower degree in the morning, and is supplanted by a reverse type in which the morning temperature is highest. Such an occurrence took place in the case reported to me by Krusen, which is quoted in Chapter I.

FIG. 12.



Abortive typhoid fever ending by the seventh day, and by crisis instead of lysis.

In this connection, too, it must be remembered that in some cases (not many), during the course of the second week, the fever develops a type closely resembling that seen in remittent malarial fever. According to many writers on diseases of children, this form of the fever is by no means rare in this class of patients. Again, as this week or the third week ends, the febrile movement may even be distinctly like that of a malarial intermittent without there being any malarial infection of the patient whatever. Strümpel speaks of such cases in which distinct remittance occurred, and of others in which the fever was completely intermittent, the afternoon temperature for two or three weeks being as

high has 104°, yet followed by morning temperatures at the normal point, and Pepper has expressed the belief that these great variations are in part the result of marked sepsis and intestinal ulceration. Thus he has seen as much as 7 degrees variation occur for several days in succession. Such variations should never be considered curiosities in typhoid fever, but should stimulate the medical attendant to increased endeavor to discover a septic source other than the intestinal lesions as, for example, a septic kidney. They may occur, however, in cases without complicating diseases or lesions, as is shown in Fig. 12.

In this man's case the blood was examined repeatedly for the malarial organism, with negative results, and there was no history of exposure to it. Cases of this type are also recorded by Herringham, who discusses these temperature variations in *St. Bartholomew's Hospital Reports* for 1896. In one of these a woman of thirty-three years had severe rigors followed by high fever on the evening of the twenty-third and the morning and evening of the twenty-fourth day of the disease. These rigors were followed by a fall of fever, which amounted to a crisis, and speedy convalescence ensued. In still another case chills and fever occurred on the thirty-first, thirty-fifth, and thirty-sixth day of the illness, followed by two attacks on the thirty-eighth day. These were in turn followed by crisis and recovery. In the other cases reported by Herringham a rigor occurred in one during the acme and later during lysis; in another at the onset of lysis; in another in lysis; in another a number of rigors occurred in acme and severe rigors in lysis, probably due to thrombosis. Osler has also reported a case of this type.[1] Church[2] has recorded a case in which a girl had twenty-two rigors in a primary attack in fourteen days, twenty-five in fifteen days in a first relapse, and six in eleven days in a second relapse.

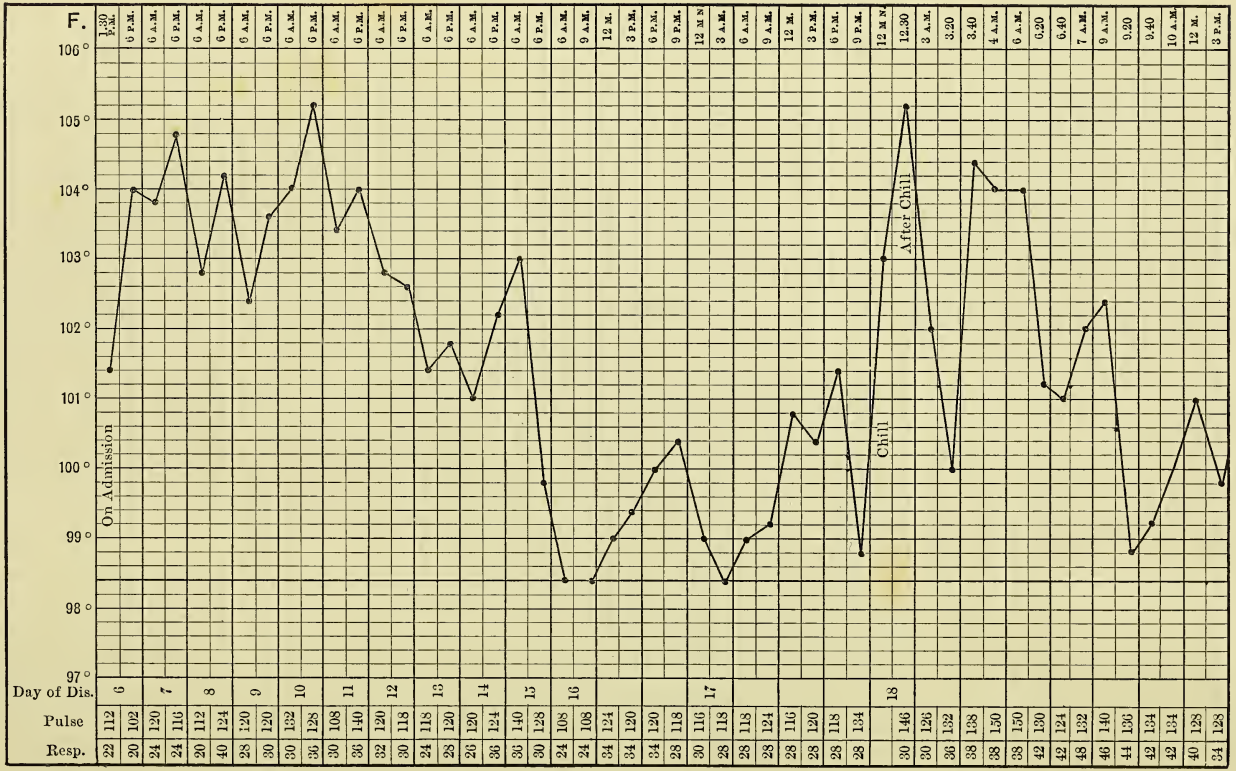
It is well to recall the fact insisted upon by no less an authority than Janeway,[3] that the use of the coal-tar products in the course

[1] Johns Hopkins Hospital Reports, 1895, No. 5.

[2] St. Bartholomew's Hospital Reports, 1896.

[3] Transactions of the Association of American Physicians, 1894.

FIG. 13.



A morning and evening chart of a case of severe typhoid fever in a large, full-bellied man. His temperature, as shown by a chart marked every two hours, was frequently as high as 105°, or more. He was treated by cold frictions. His fever, as shown in this chart, seemed about to end by crisis when he was seized with severe chills, two of which occurred on the eighteenth, one on the nineteenth, and one on the twentieth day of his illness.

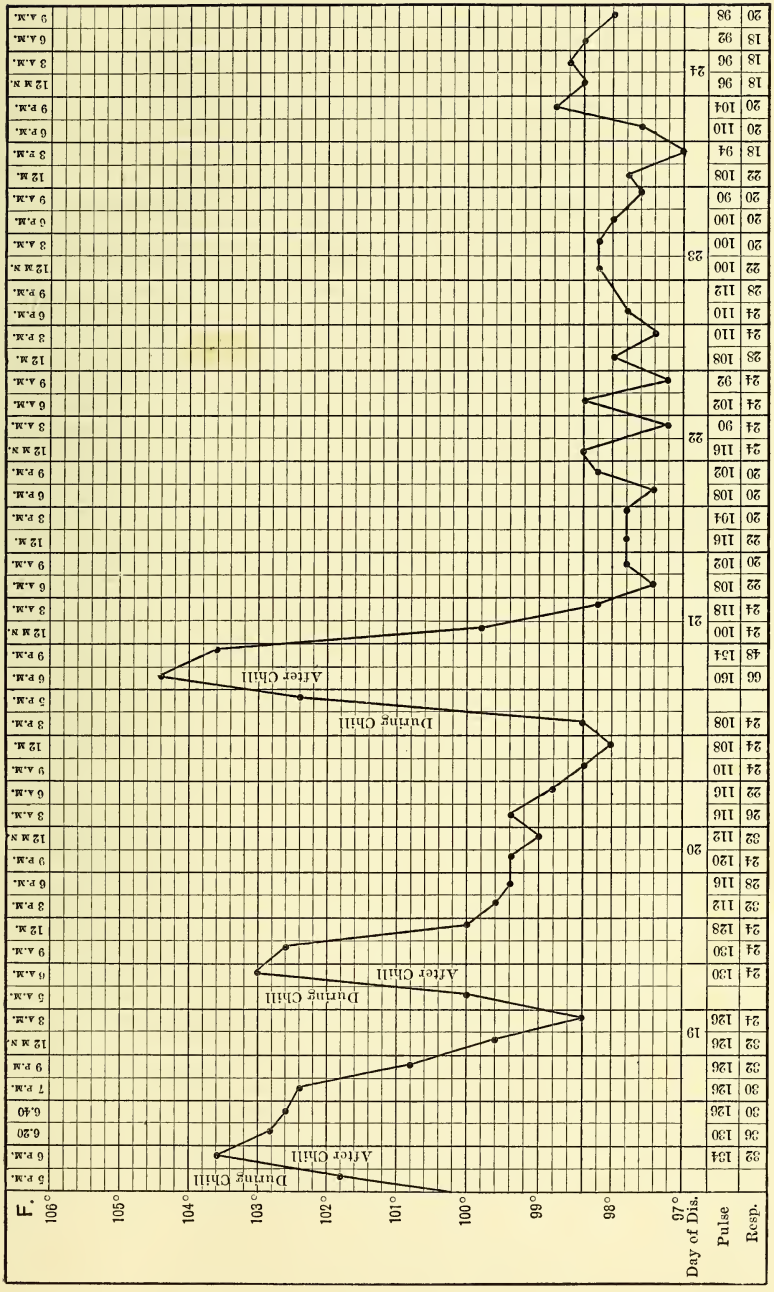


FIG. 13 CONTINUED.

of enteric fever may have a chill-producing effect. It is well known that the external use of guaiacol will produce severe rigors.

In other cases presenting such rigors there is present a true double infection of typhoid and malarial fever. (See further on.)

There are a number of conditions which result in producing a marked and sudden fall of temperature during the periods of the fastigium and defervescence aside from the sudden drop, rarely seen, in which the fever ends by crisis instead of lysis, the patient passing into convalescence at once. The most important of these causes, both because of their degree and because of what they indicate, are hemorrhage from the bowel, or, if it be profuse, that from any other part of the body, perforation of the bowel and the rigor preceding a complicating infection such as pneumonia, the beginning of a relapse or the effect of powerful antipyretic drugs. Often great falls in temperature take place when the typhoid infection is associated with malarial infection, as already intimated. (See further on.)

In the case of a complicating disease a few hours' delay in recognizing its presence may not make much difference to the physician or patient; but, on the other hand, the early recognition of hemorrhage or perforation may save the patient's life. The symptoms of perforation, associated with the fall of fever, are prominent and will be considered under the head of gastro-intestinal accidents, but in the case of intestinal hemorrhage the fall may occur some time, it may be several hours, before the appearance of a bloody stool enforces the belief upon the nurse that hemorrhage is present. For this reason an unexplained marked fall of temperature should always be regarded with suspicion, and the appearance of the next stool watched with interest. The pulse should be carefully studied for signs of loss of blood, and the facial expression and color of the tongue closely watched. If the patient is conscious and capable of giving expression to his sensations he may complain of a sensation of faintness or of sinking, or if the hemorrhage is very profuse the patient may pass rapidly into a state of collapse or shock, owing to the extravasation of blood into the small and large bowel, dying almost simultaneously

with the gush of blood from the rectum. Thus I have seen a case apparently passing safely through a moderately severe attack of enteric fever suddenly develop the symptoms named, present all signs of marked exsanguination, and then pass into the bed an enormous volume of half-clotted blood, which extended from the anus to the heels, at the same moment developing gasping respiration, profound syncope, and seeming to be *in articulo mortis*. So, too, I have seen actively employed hypodermoclysis result in the recovery of patients so greatly exsanguinated that death seemed inevitable.

Sometimes, however, even profuse intestinal hemorrhage recurring again and again, fails to cause a very great fall in the temperature, or does not keep it low but for a short time.

Sometimes well-developed signs of collapse appear in the course of typhoid fever without indicating any serious accident in the course of the disease which could produce these symptoms. In this state the patient develops a rapid pulse, shallow respirations, pallor and lividity, accompanied it may be by a rigor. There is usually a marked fall of temperature. Herringham[1] asserts that these symptoms have no effect on the prognosis, and that treatment is practically unavailing. On the other hand, they may mean that the patient is in grave danger, as has been pointed out by Landouzy and Siredey.[2] (See circulatory changes in the well-developed and convalescing stages of the disease.)

How far constant fever occurring day after day and associated with manifestations of general loss of strength and debility can be relied upon in the diagnosis of typhoid fever is hard to determine. Certain it is that if a physician makes a diagnosis of enteric fever upon these symptoms alone, without bearing in mind the fact that similar conditions are equally well developed under other forms of infection, he will find himself in error in not a few instances. Chief among these may be mentioned tuberculosis of the lungs or peritoneum, that form of influenza in which the chief

[1] St. Bartholomew's Hospital Reports, 1896.

[2] Revue de Médicine, 1887, p. 804.

symptoms are abdominal, cases of ulcerative endocarditis, septicæmia and pyæmia, and those of cholecystitis with ulceration, as from impacted gallstones. It must not be forgotten, too, that syphilitic fever may in very susceptible persons resemble typhoid infection. The febrile movement, rose rash, if it be scanty, malaise, and signs of general infection may readily mislead the physician.

Again, in the more advanced stage (tertiary) of syphilis prolonged, low septic fever may be present.

Finally, let it not be forgotten that trichiniasis[1] may resemble typhoid fever, for in it we have fever, pains in the limbs and back, headache, stupor, and nausea, with pain in the belly and diarrhœa.

Points in differential diagnosis in this condition are the presence of leucocytosis (particularly in eosinophiles), and its absence in typhoid fever, and puffiness of the bridge of the nose.

Not only may the fever of these states be moderate and prolonged and the evidences of asthenia marked, but enlargement of the spleen, diarrhœa, and tympanites may be present. The difficulties in differential diagnosis in cases of suspected gall-bladder disease are increased by the fact that such disease often has its origin in an old infection of the gall-bladder due to an attack of typhoid fever months or years before, the bacillus of Eberth being present in this viscus during the entire interval, or in other cases it invades the gall-bladder at the onset of the infection of the entire body, and so emphasizes the hepatic symptoms. Further than this, cases which have previously had enteric fever may also give the Widal test, although the immediate cause of the attack may be localized in the manner named. These forms of infection will be considered later on.

Reference has already been made to the possibility of the febrile movement resembling that of malarial fever. In some cases this infection is truly present, but in others the temperature-chart is that of an irregular typhoid fever.

These facts bring us face to face with a discussion of a subject

[1] As the most recent paper on this subject, see Osler, American Journal of the Medical Sciences, March, 1899.

about which great diversity of opinion exists, and has existed for years, namely, the question of that condition which has been called "typho-malarial fever." At the present time it may be asserted as a fact that a separate disease entity of this character does not exist, and this is done on the basis that recent discoveries in the natural history of these diseases, particularly the recognition of the malarial germ on the one hand and the use of the Widal test on the other has enabled us to make an absolute diagnosis in cases in which so positive a statement has heretofore been impossible.

There is no doubt whatever that pure typhoid infection may result in the production of a fever which closely follows the remittent and intermittent malarial types, and which is often associated with so much gastric disturbance and vomiting and so lacking in the more prominent typhoid symptoms usually seen that the picture of remittent malarial fever is clear, while the true picture of typhoid fever is clouded. (See also chapter on diseases which ape typhoid fever.) Again, there can be no doubt that cases of true malarial infection occur in which the symptoms so closely resemble those of typhoid fever that a purely clinical diagnosis is almost impossible, particularly if an epidemic of typhoid fever is in full swing at the time. Finally, there can also be no doubt that it is possible for the patient to have a double infection with the bacillus of Eberth and the plasmodium of Laveran, in which case, however, the malarial manifestations are usually dwarfed by the typhoid poison, and only are marked at the onset of the enteric fever and at its termination. To this mixed infection the term typho-malarial fever may be correctly applied to indicate not a separate disease, but a double infection. Etymologically, this term might also be used to define a condition of malarial fever in which, because of profound debility, the patient was in a typhoid state—that is, in a condition of which typhoid fever is a type. Practically, however, it should be discarded or limited in its use to the double infection just described.

Johnston has well said, "As at the present employed the term typho-malarial fever has no determined meaning, leads to

confusion and misunderstanding, is a cover for uncertainty and ignorance, and should be discouraged and abandoned."

As already shown, there can be no doubt that mild grades of typhoid infection take place in which the only symptom of this disease is fever which runs a moderate course, and is accompanied by a certain degree of general debility. Often they begin rather abruptly, with a slight chill, or gradually the patient feels less and less well till he takes to his bed. These cases are characterized by well-marked remissions, it may be, and suffer from somewhat indefinite symptoms difficult of classification. They do not respond to quinine, nor do they show any typhoid symptoms other than those named, and the diagnosis arrived at will depend largely upon whether the physician is practising in the North or the South, is treating many cases of enteric fever or many of remittent fever, unless he is skilful with his microscope, in which case the Widal reaction for typhoid fever in a majority of cases will at some time settle the diagnosis for him, or an autopsy will show typhoid lesions.

Or, on the other hand, he may find the malarial organism in the blood, which will prove that this infection is present, although it will not exclude typhoid fever, just as the Widal test will not exclude malarial infection.

Atkinson has well described that form of typhoid fever resembling malarial fever of the remittent type in the following words:

"From beginning to end the patient may develop no symptom that could not belong to this disorder (malarial fever), except the persistence of fever under strongly antimalarial treatment and the occasional occurrence of circumstances that point to a typhoid origin. There is no intellectual cloudiness or hebetude of expression. Sleep is but slightly disturbed. The tongue remains moist and coated with a thin whitish or yellowish fur; the appetite persists very often in some degree. There is almost never epistaxis. Constipation is commonly observed, diarrhœa very rarely. There are no bloody stools, no tympanites, no iliac tenderness or gurgling. Rose spots are much more often absent than present. The patient can be restrained in bed with difficulty or under protest. Slight

enlargement of the spleen may occasionally be detected, but is more frequently not observed. More severe cases, beginning more or less abruptly, develop primarily the symptoms of remittent fever, and diagnostic doubts only arise when the absolute resistance to anti-periodic treatment and the gradual appearance of typhoid symptoms excite suspicions of the incorrectness of the original diagnosis."

(For a description of infectious processes complicating typhoid fever, see further on.)

The Course of the Fever in Relation to Prognosis. It has already been pointed out that fever of sudden onset, soon followed by a fall or affected by marked remissions during the stage of onset, is a favorable rather than an unfavorable omen. A somewhat similar statement holds true in regard to the fever of the well-developed disease in which the presence of persistently high morning and evening temperature, the variation between the two being but slight, possesses an evil significance, while, on the other hand, marked differences between these points are considered of good omen. This is so because remissions indicate that the fever is not violent and because remissions permit the body to make repairs to enable it to stand another rise, whereas the constant maintenance of high fever seriously impairs the vitality of the tissues. This temporary reduction of fever is probably one of the ways in which the cold bath does good.

In regard to the prognostic value of high temperatures we find considerable unanimity of opinion. Liebermeister, in studying 400 cases, found that of those whose temperatures rose to 104° or more 9.6 per cent. died; of those whose fever exceeded this degree, 29.1 per cent. died, and of those whose axillary temperature exceeded 105.8°, more than half died. Fiedler[1] found that when the temperature reached 106° more than half died, and Wunderlich states that at 106.1° the danger is considerable, at 107° the deaths are almost twice as numerous as the recoveries, and at 107.2° and over recovery is rare. Concerning the influence

[1] Deutsches Arch. für klin. Medicin, Bd. i. p. 534.

of high morning temperatures, Fiedler says that practically all patients died whose morning fever rose to 106.2°, and that more than half died if their morning fever reached, if only once, 105.4°.

In the Maidstone[1] epidemic only one death occurred in 81 cases, the temperature of which reached less than 104°, whereas nine deaths occurred in patients who had fever at some time above 104°, but a case is recorded of recovery after a temperature of 110°.[2]

While acute hyperpyrexia is an evil omen in enteric fever, long-continued, moderately high fever is, perhaps, more harmful. In the Boylston Prize Essay of Harvard University for 1890 the writer used these words in speaking of this subject:

"Closely allied to this question of hyperpyrexia is that which asks us to define what we mean by hyperpyrexia. As given in most works on fever, this term is applied to any state in which the temperature reaches 106° or 107° F.; but in reality the figures have little to do, except in an indirect way, with what student or physician wishes to know. A temperature of 106° F. in a young healthy man suffering from an acute attack of some short-lived disease does not mean very great danger; but a temperature of 103°, day after day in typhoid fever, does mean danger, and must be carefully attended to. In simple, continued fever 106° F. is a hyperpyrexia; in typhoid, or other low fever, 103° F. is a hyperpyrexia. The question is not one of actual degrees Fahrenheit, but rather as to whether the temperature present is doing any harm."

Very great differences are to be found in different patients in respect to the persistency of high fever under the application of hydrotherapy. In some instances active bathing serves to reduce the fever but slightly; in others moderate measures produce a marked effect. As an illustration of the great fall produced by sponging with ice-water for twenty minutes, with active friction,

[1] Poole. Guy's Hospital Reports, 1898. Wrongly labelled on cover, 1896.
[2] St. Thomas's Hospital Reports, 1895, p. 248.

reference may be had to the following chart (Fig. 14), in which it is seen that as great a fall as 8° F. occurred. One is tempted to inquire how low it would have fallen had the routine method of plunging every patient sick with typhoid fever been instituted. Yet the patient was an unusually heavily built, stalwart lad of twenty years, well nourished, and in good condition for bathing. Further, he came under care by the third day of his illness.

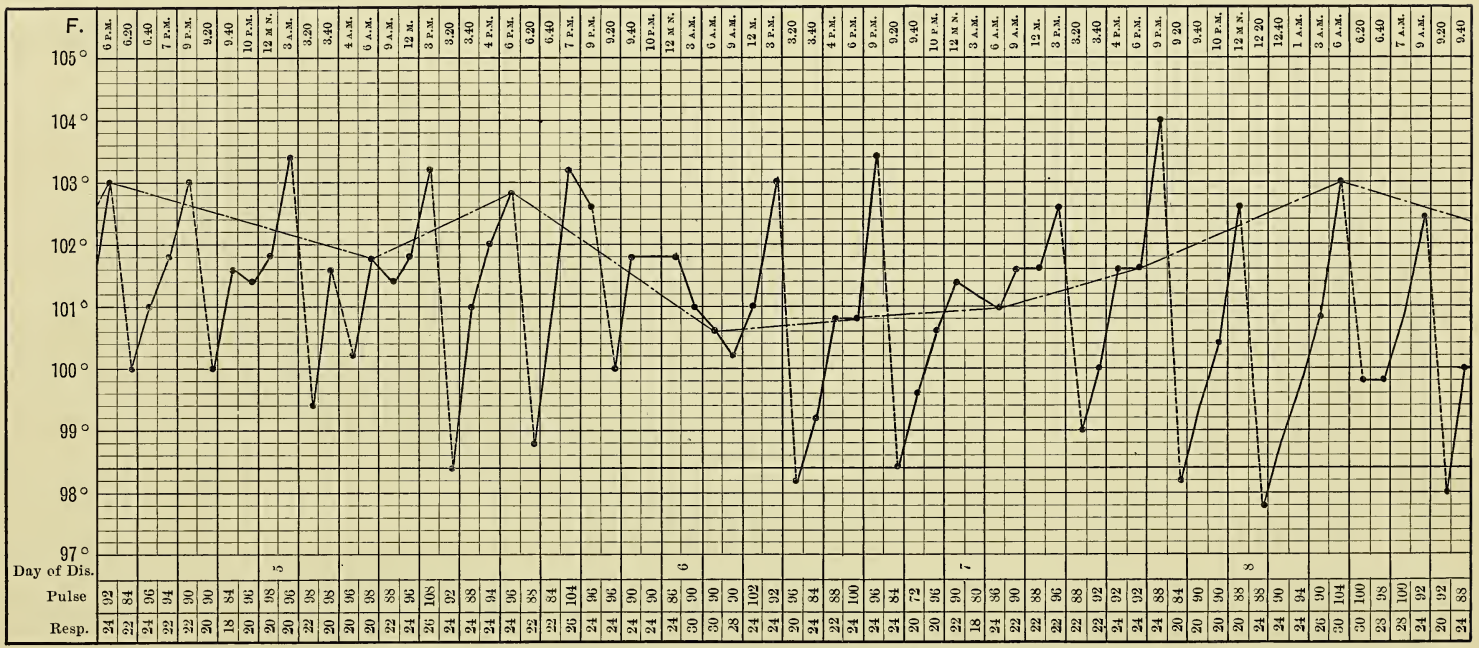
Respiratory System in the Developed Stage of the Disease. The respiratory functions of patients suffering from typhoid fever are not materially disturbed unless some complicating affection of the lungs or nearby organs develop. Beyond a slight quickening of the respirations, varying from two to eight a minute, as the result of the fever, they maintain an even rhythm. The development, therefore, of rapid or noisy breathing is indicative of some pulmonary, cardiac, or renal complication, and deserves close scrutiny and study.

Before discussing the graver respiratory complications of this malady, there are, however, several minor facts in connection with this part of the body which deserve notice. One of the first of these is the curious fact that coryza is almost never met with in typhoid fever in any of its stages, and its presence with other signs pointing to enteric fever stands against the presence of this malady.

Another point of interest is the frequency of epistaxis, which is chiefly met with in the first week of the disease, as already pointed out, and which is also seen quite commonly later on, probably being produced in most instances by the patient picking the nose to remove crusts, while in the early stages it is a means that the system takes for relieving the frontal headache and congestion which are so common at that time. J. M. Da Costa[1] presented in a recent clinic two patients who had this symptom late in the disease. The first patient had been ill twenty-nine days, and his temperature had reached normal. The bleeding was violent, lasting half an hour, and several ounces of blood were lost. Cerebral

[1] Medical Fortnightly, February 1, 1899.

FIG. 14.



This chart shows the primary resistance to cold in the first week of the disease, and the gradually increasing response to cold as the disease progressed. The second page shows the great falls of temperature caused by cold frictions, and the third page the fact that, in this patient at least, tepid sponging with friction was sufficient to control the fever. The dotted line is the fall of fever under treatment, and occurred after twenty minutes' use of cold. The next solid line shows the temperature twenty minutes later. The broken line is the morning and evening range.

FIG. 14 CONTINUED.

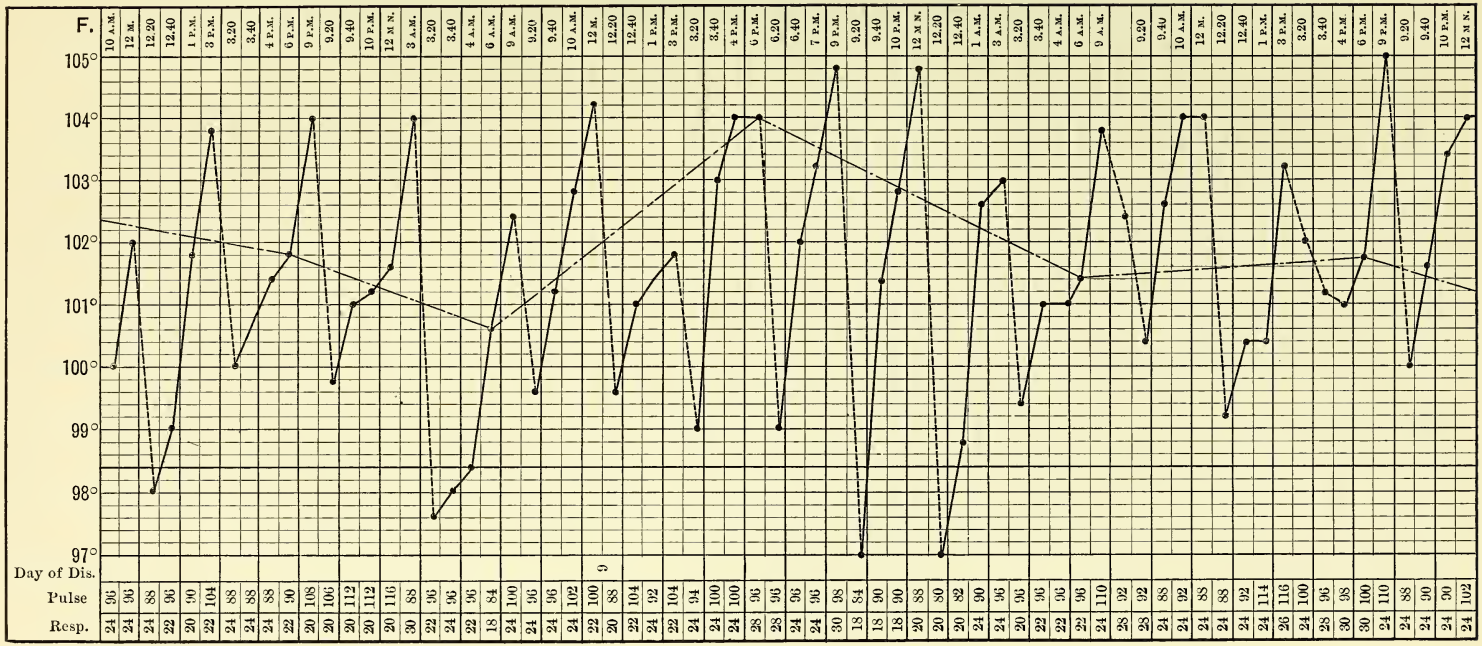
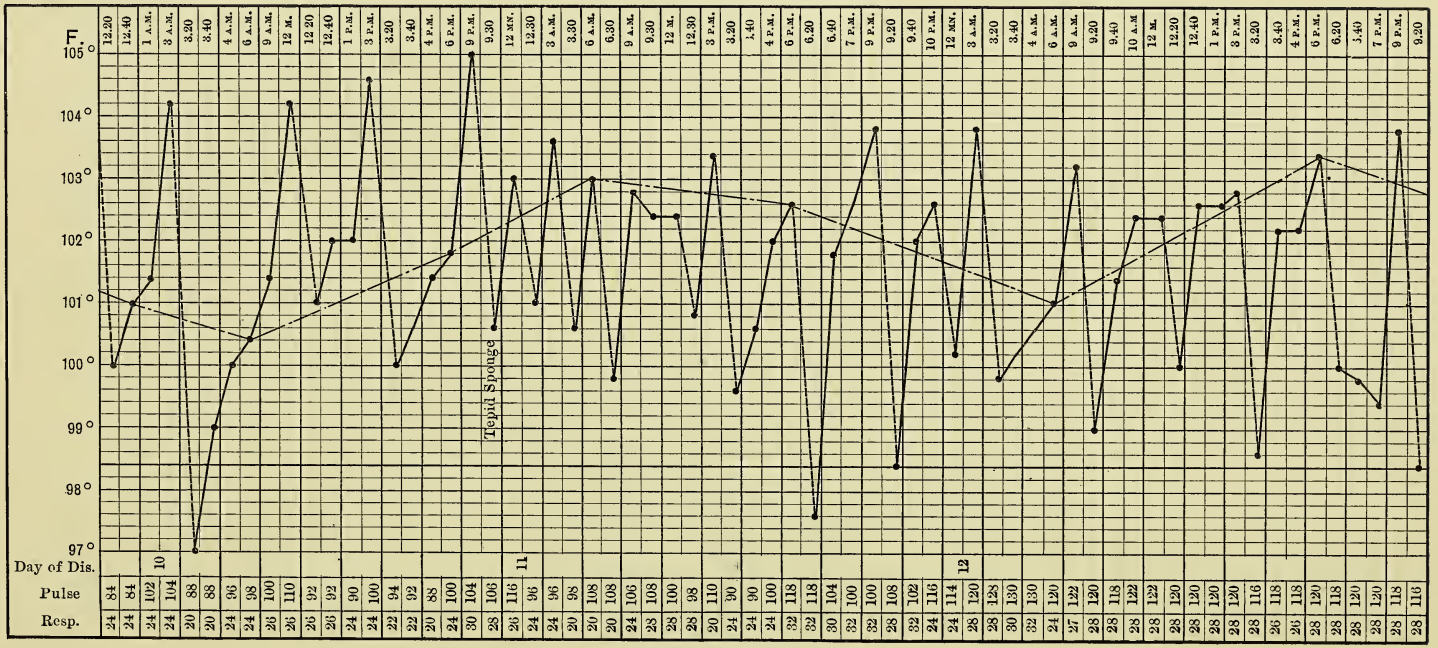


FIG. 14 CONTINUED.



symptoms were relieved, and the man made good progress afterward. The second patient had profuse bleeding during the fourth week of the disease, after symptoms of typhoid fever had practically ceased. Late epistaxis is more apt to occur, in Da Costa's opinion, after severe cerebral symptoms, which are thus relieved. In still other cases the hemorrhage from the nose is part of the manifestation of a general hemorrhagic diathesis. Very rarely is the symptom excessive enough to require active interference, and still more rarely does it cause death. Thus out of 1420 cases seen by Liebermeister, epistaxis took place in 107 cases, but death occurred from this cause in only two, and this is probably a high percentage.

Perichondritis of the larynx complicating typhoid fever, occurs in less than 1 per cent. of all fatal cases, and Trousseau has pointed out that it is most apt to occur when the patient suffers from profound exhaustion, particularly if his attack has been a prolonged one. Schultz, who analyzed 4094 cases of typhoid fever which occurred in Hamburg in 1886 and 1887, does not record any cases of perichondritis, and Jacob does not mention this complication. That this accident may be due to the local action of the bacillus of Eberth seems to be very probable, and Lucatello[1] believes that he has proof that it is the cause of the affection. On the other hand, Dittrich[2] asserts that the process is due to the dorsal position of the patient, and is more directly the result of the pressure of the laryngeal cartilages, particularly the cricoid rings on the vertebral column. By this means their vitality is impaired and their invasion by pyogenic micro-organisms is rendered easy. Lemcke[3] records a case of this affection occurring in a Swede aged twenty-four years. A similar case has been reported by Tooth,[4] in which a boy of five years suffered from typhoid fever and developed on the eighteenth day of his illness

[1] Beitrag zür Pathogenese der Kehlkopaffectionen Beim Typhus. Berliner klin. Woch., 1894, vol. xxxi. p. 379.

[2] Handbuch dür Spec. Path. und Ther., Bd. i. p. 311.

[3] Chicago Medical Recorder for 1897, vol. ii. p. 114.

[4] London Lancet, April 2, 1893.

laryngeal cough and aphonia. Tracheotomy was performed without relief. The cervical glands were enlarged and death finally occurred. At the autopsy the larynx and trachea were found lined with diphtheritic membrane. Whether this was due to the bacillus of Loeffler or to the bacillus of Eberth is not stated. Finally, Eppinger[1] believes that the ulcers which form in the larynx in typhoid fever are slightly analogous to the ulcers which form in the intestines, since he has discovered the bacillus of typhoid fever in these ulcers.

In an inaugural thesis upon ulcerations of the larynx in typhoid fever, Grüder[2] describes three types of this disease. In one of these there are specific ulcers occurring simultaneously with those in the bowel, although the bacteriological examinations failed to show the presence of the specific bacillus in these ulcers. In the second class there are simple catarrhal manifestations with a tendency to ulceration. Both of these classes involve the posterior wall of the larynx on the ary-epiglottic fold. The third class is that in which ulcers formed at the margin of the epiglottis. These usually occur singly.

Laryngeal ulceration occurs in a fairly large proportion of the severe cases, and is usually due to secondary infiltration of the laryngeal mucous membrane, apart from true typhoid infection, arising from the general debility of the patient. Usually these ulcers form at the posterior part of the larynx, and often involve the insertion of the vocal bands. Under these circumstances they may cause hoarseness and aphonia, but often they exist if in moderate degree, with but little discomfort to the patient. Rarely a painful laryngeal cough develops, and if they extend to the epiglottis they may cause pain in swallowing. Contrary to what might be supposed, they rarely lead to serious difficulty, nor do they materially affect the course of the disease. Very rarely they produce perichondritis of the larynx or œdema of the glottis. Hoffmann found laryngeal ulcers in twenty-eight cases out of 250

[1] Ziegler. Path. Anatomie, Bd. ii. p. 626.

[2] Centralblatt f. Bacter. und Parasit., February 17, 1891.

typhoid autopsies, and from his studies it is evident that this lesion may occur in the second week of the disease. Griesinger found them in 26 per cent. of the cases that died, and that the lesion is more common in men than in women. These statistics show that in severe cases of typhoid fever resulting in death the laryngeal lesions are more commonly present than is generally thought, and illustrate the fact already pointed out that unless the ulceration is widespread and the ulcers involve the epiglottis and vocal bands, no marked symptoms of laryngeal trouble may present themselves. On the other hand, in 166 cases of typhoid fever Landgraf[1] found laryngeal complications to be rarely present; in some instances they had apparently been present during the early stages of the disease, but had healed before death occurred. Only three cases of perichondritis and two cases of muscular paralysis were met with, the latter during convalescence.

(For a discussion of laryngeal paralysis see the chapter on the stage of convalescence.)

An interesting case of so-called laryngo-typhus has, however, been recorded by Lewy[2] as occurring in a child of one year; death occurred on the eighth day, and the autopsy, in addition to revealing the intestinal lesions of typhoid fever, also showed fibrinous laryngitis and croupous pneumonia. A case of necrosis of the two arytenoid cartilages has also been reported as occurring in a man, aged eighteen years, by Souques.[3]

When severe laryngeal disease asserts itself the condition of the patient is apt to become at least pitiable, and it may be alarming. The largest number of cases collected of this affection are those of Lüning,[4] who, in 1884 collected 213, although Keen, in 1876, had collected 169 cases. Westcott, in collecting statistics for Keen's well-known monograph on the *Surgical Complications of Typhoid Fever*, collected thirty-eight others. Basing his views upon his statistics, and in particular upon fourteen original

[1] Deutscher Medicinischer Wochenschrift, January 6, 1890.

[2] Archiv für Kinderheilkunde, Bd. lx., heft. 3, 1888.

[3] Bulletin de la Société Anatomique.

[4] Archiv für klin. Chirurgie, 1884, vol. xxx. p. 225.

cases, Lüning gives the following graphic word-picture of the conditon :

"Physician and patient together rejoice over the daily progress toward convalescence ; of the still slight but persistent trouble in the throat, scarcely a word is said, until all at once—an exposure to cold, a little walk, is then usually blamed for it—the hoarseness increases, and swallowing becomes markedly painful. The picture now quickly alters. Soon, often within a few hours, come dyspnœa and suffocating attacks. Sometimes even during the very first day the anxious scene of laryngeal stenosis sets in, with stridor, inspiratory depression of the neck and chest-wall—the unrest of despair, a struggle with death. The face becomes livid ; the respiration becomes rapid, wearisome ; the auxiliary muscles of respiration are all called into play ; sometimes the respirations are prolonged and noisy. The patient can find no rest ; the dyspnœa even prevents the taking of nourishment ; the expectoration of the increasing mucus becomes imperfect ; soon attacks of suffocation recur. Either a tracheotomy must now be done immediately, or the patient, if he is weak, may choke to death, even in the first attack. More commonly, however, the attack subsides, and a slight improvement with a short sleep will ensue. Expectoration of bloody mucus, masses of pus, and, in some cases, even of pieces of cartilage, diminish the symptoms, and show at the same time that the real cause of the dyspnœa is not a catarrhal œdema or dropsical swelling, but a destructive ulceration, even of the cartilages. Often, also, there is severe fever. Thus pass on, it may be, even days and weeks, easy breathing alternating with the suffocative attacks. The alternative is only a finally fatal attack of suffocation, or a late palliative tracheotomy with all its uncertainties. . . . If one will read the cases of death from suffocation without operation (52 cases, 49 deaths), he will find that, almost without exception, suffocation occurred early and quickly, before either physician or patient had even thought of tracheotomy.

"This is the picture in cases of perichondritis. If the patient is in the stage of typhoid stupor, when the ulceration is accompa-

nied with acute suppuration and swelling which may lead to destruction of the cartilages, the initial symptoms of the threatening danger may escape us entirely in spite of careful observation. . . . In these cases the objective signs of laryngeal stenosis, on which we usually depend, are much less marked; stridor, movements of the larynx, inspiratory depression, action of the auxiliary inspiratory muscles—in short, everything by which, in the healthy, we make the diagnosis of narrowing of the air-passages is, in the *vita minima* of the weakened patient, far less outspoken, and easily deceives us as to the degree of the danger of suffocation. The striking suffocative attacks, with arrest of respiration, so alarming even to the lay observer, are less noticeable, since the struggle of the patient with the mechanical obstruction quickly fails or is quickly abandoned. The condition passes into a death agony with œdema of the lungs, without the stenosis seeming to have reached a threatening degree. . . . And thus one sees, often with astonishment, in the reports of the necropsies, how often the stenosis and destruction of the cartilages occur, as it were, 'without even any symptoms.'"

Lüning's statistics seem to show that severe laryngeal ulceration is far more frequent in Germany than in England or America, and in the latter country it must be very rare.

Keen's essay points out that emphysema and suppuration of the mediastinum may follow perforative ulceration of the larynx, and Wilks[1] records the case of a patient of twelve years, who on the twelfth day of the disease developed general emphysema due to this cause. Denham[2] records a similar case in a boy of ten years, and Chomel[3] another in a man of twenty years, from a perforation of the thyroid cartilage. One instance is recorded by Lüning in which an abscess had destroyed the arytenoids and rendered the cricoid necrotic, so that the anterior mediastinum was filled with pus, and Retslay[4] records another in which a

[1] Medical Times and Gazette, 1862, vol. ii. p. 276.

[2] Holmes' System of Surgery, 2d, ed., vol. iv. p. 571.

[3] Thèse de Paris, 1877.

[4] Retslay, Ueber Perichrondritis Laryngéa Berlin, Dissert. 1870, No. 10.

perichondral abscess about the thyroid cartilage caused secondary involvement of the anterior and posterior mediastinum.

Keen's table shows that in 146 cases of severe laryngeal disease 12 occurred under fifteen years, 87 between fifteen and twenty-five years, and 47 over twenty-five years.

The marked exemption of children is evidently associated with the mild character of the disease in this class of patients. Lüning's table of 165 cases showed 18 under fifteen years, 109 between fifteen and twenty-five years, and 28 between twenty-five and thirty years, and 10 between thirty and thirty-five years or over, giving results of a similar character. The far greater frequency of the malady in men than in women is interesting, for in the female the general disease is as severe as in males, as a rule, yet in Keen's table there were 119 males to 29 females, and in Lüning's table 129 males to 36 females. Keen tells us in regard to the date of onset that 7 cases occurred in the first week, 23 in the second, 30 in the third, and 82 in the fourth week to two months.

Keen states that necrosis of the cartilages is by far the most common and also by far the most dangerous form of laryngeal affection, but adduces no evidence in support of its being the most common lesion. Opposed to this view we have that of Liebermeister, who tells us that "laryngeal ulcers do not in any way affect the ordinary course of the disease, and in favorable cases heal without leaving any evil consequences." "Occasionally," he tells us, "they may lead to death by producing perichondritis laryngea or glottic œdema." This difference of opinion rests upon a difference in the severity of the lesions. Surgeons only meet with cases which are severe enough to demand operative relief, whereas physicians comparatively commonly see the milder forms. When necrosis of the cartilage does take place there can be no doubt that Keen's statement as to the danger being great is correct, for in this condition his statistics show that the mortality approximates 95 per cent. In 197 cases of laryngeal stenosis in enteric fever Keen records a mortality of 67 per cent., which if the cases are divided into those operated

on by tracheotomy equals 55.5 per cent., and not operated on, 78.6 per cent. That operation is imperative as soon as suffocative attacks are threatened, is evident.

The *bronchitis* of advanced typhoid fever is a very constant symptom, so constant that it really forms part of the symptom-complex of the regular disease. It is only when it becomes severe and passes into a broncho-pneumonia that it possesses any considerable interest, for if at all well developed it becomes a grave menace to the patient's life. This lobular pneumonia depends upon four separate causes for its existence. First, the bronchial irritation characteristic of the disease; second, the feeble respiratory movements of the patient, and the dorsal decubitus whereby dependent portions of the lung collapse; third, the feeble circulation which permits stasis in the pulmonary vessels; and, finally, and very important, the inspiration into the lungs of particles of food or foreign bodies in the mouth or nose which are septic, or which decompose, and produce pneumonia in this manner. The physical signs of this form of the diesase are identical with those of ordinary lobular pneumonia, and the prognosis is bad in direct proportion to the feebleness of the heart and general system, the extent of the lesion, and the slowness with which the heart and general system responds to stimulation. Hoffmann tells us that this complication was found 38 times in 250 autopsies; so it is evident that its influence in producing a fatal result is probably not very great, as a rule. It is emphatically a symptom pertaining to feeble and debilitated patients, and most often comes on in the latter part of the second or third week. As is often the case lobular pneumonia may afford a favorable field for the growth of the bacillus tuberculosis, and, therefore, in those cases in which resolution does not take place, pulmonary phthisis not infrequently follows this form of the disease. Mettenheimer[1] saw thirteen cases of this character out of thirty-eight deaths from typhoid fever or its sequelæ.

[1] Beobachtungen ueber die typhoiden Erkrankungen der franzosischen Konigsgefangenen in Schwerin, Berlin, 1879.

Very much more rarely acute miliary tuberculosis develops in typhoid fever, probably because the focus of some earlier and dormant tubercular infection breaks down and sets free tubercle bacilli in a system the vitality of which is depressed. Hoffmann found it four times in 250 typhoid fever autopsies.

Hypostatic congestion of the lungs, a condition closely allied in causation and prognosis to lobular pneumonia, occurred in 100 out of 1420 cases recorded by Liebermeister, and pulmonary œdema is the usual immediate cause of death in cases which die of failure of the cardiac muscle, as Hoffmann has proved.

True croupous pneumonia occurring in the later stages of typhoid fever, either as a result of an infection with the micrococcus lanceolatus or by the bacillus of Eberth, is a very rare affection, much more rare than it is in the stage of onset as already pointed out. Hoffmann found it present only eighteen times in 250 typhoid autopsies. Again, in 1420 cases quoted by Liebermeister, 52 cases had "extensive consolidation" of the lung not dependent on hypostatic congestion. A "good many" of these, however, were probably cases of true lobular pneumonia and were not croupous.

In this connection it is interesting to note that as long ago as 1839 Becquerel wrote an article on pneumonia complicating typhoid fever when making an analysis of eighteen cases in the service of Jadelot in 1837.

Hemorrhagic infarction of the lungs arises in typhoid fever from several causes, and is usually met with in cases with greatly impaired circulation. It is due to emboli arising in the right side of the heart or, very rarely, to emboli arising from a phlebitis. (See circulation in convalscence.)

It has been suggested that it may arise, when septic, from the intestinal ulcers, but no case of this kind has come to my notice.

Sometimes it may arise from a bed-sore, a parotid abscess, or or from an abscess elsewhere.

In many cases the presence of small infarctions is unsuspected, either because they cause little difficulty or because they are not differentiated from lobular pneumonia, the physical signs in each case being nearly identical. When the infarction is large we have

a rise of temperature, pain in the chest, currant-jelly blood in the sputum and, if the embolus is septic and the patient survives signs of pulmonary abscess or gangrene. Sometimes the infarction is due to thrombosis. The presence of a focus which can supply an embolus and of a feeble heart, increase the probability of the pulmonary difficulty being infarction, and an infarction severe enough to be recognized is of evil prognostic omen. Out of 250 typhoid autopsies Hoffmann found fifteen cases of hemorrhagic pulmonary infarction.

Hæmoptysis complicating typhoid fever in a patient free from tuberculosis may occur. Creagh[1] has reported such an instance in a man of thirty-five years; the accident resulted in death. Unfortunately, no autopsy was made in this case to prove that there was no local tubercular lesion; but it is possible that such hemorrhages may occur without tuberculosis.

Primary pleurisy complicating typhoid fever is very rare. Nearly always it is secondary to infarction, pneumonia, or gangrene. Rarely it may be due to direct typhoid infection, and when this is the case the effusion is usually purulent. As early as 1885 Rendu and de Gennes,[2] and in 1887 A. Fraenkel[3] obtained the bacillus of Eberth from the pus of an empyema. In Keen's essay Westcott has collected nine instances of typhoid pleural effusion, in five of which this specific organism was found. As a rule, this state comes on as a late symptom, not earlier than the third week, or sometimes not until two months after the fever.

Further, in support of the statement as to the secondary character of pleurisy, out of these nine cases it succeeded pneumothorax once,[4] pulmonary abscess once,[5] gangrene of the lung once,[6] and suppurative mediastinitis once.[7]

[1] London Lancet, November 30, 1895.
[2] La France Méd., 1885, vol. ii. p. 1821.
[3] Verhandlungen Sechste Kongress für Inner. Med., 1887, p. 179.
[4] Rendu. La France Médicale, 1885, vol. ii. p. 1809.
[5] Ramsey. Annals of Surgery, January, 1890, p. 39.
[6] Griesinger. Infectionskrankheiten.
[7] Barr. Liverpool Medico-Chirurgical Journal, 1893, vol. xiii. p. 346.

The prognosis is apparently very good, as six of these nine cases recovered after aspiration or drainage, including that with pus in the mediastinum.

Empyema due to the streptococcus, occurring in the course of typhoid fever, is also reported by Hanquet.[1]

A case of empyema complicating relapse in typhoid fever, in the pus of which typhoid bacilli were found in large numbers, has been recorded by Valentine.[2]

A case of gangrene of the lung in a boy of eight years, occurring as a sequel to typoid fever has been recorded by Acker.[3] Death occurred.

Circulation in the Developed Stage of the Disease. The development of fever in enteric infection is accompanied by an acceleration of the pulse-rate, as it is in all maladies. With the onset of the disease the heart, not yet weakened by illness, may not only greatly quicken its beat, but also cause the pulse to be more strong than normal. As the disease progresses, however, the pulse becomes weaker and weaker in severe cases, and the heart-sounds more and more feeble till they may be inaudible even with the most careful auscultation. With the ordinary quickening of the pulse and its common alterations we have little to do at this point. The points that interest us are the unusual variations, which consist chiefly in dicrotism, tachycardia, bradycardia, and intermittence, relaxation of the vascular pathways on the one hand, and aberrant action of the heart as to force and sounds on the other. Dicrotism may be present for days at a time in feeble cases, and is an unfavorable sign of not great gravity unless associated with other grave symptoms. Ordinarily pulse-rates varying between 80 to 120 can be regarded by the physician with equanimity, although much depends upon the character of the pulse, and still more upon the quality of the heart-sounds, which should always be studied in connection with the pulse. With each ten additional beats the gravity of the

[1] Archives Médicale Belges, June, 1892.

[2] Berliner klin. Wochenschrift, 1889, No. 15.

[3] Archives of Pediatrics, September, 1896.

condition greatly increases, and if a pulse rises to 140 or 150 per minute without some momentary exciting cause, and remains so rapid, the condition is indicative of doubtful recovery. If at the same time there is coldness of the extremities, independent of contact with ice-bags or other extraneous causes, dissolution may be imminent. Much depends, however, upon the quality of the pulse-wave. If it is full and possesses an approximately normal tension, the danger is less grave than if it is gaseous and relaxed and easily extinguished. Sometimes auscultation of the heart will show that it is acting strongly yet pumping futilely in an attempt to fill relaxed and dilated vessels.

It has been asserted by some clinicians that much prognostic information can be gained from the heart-sounds in typhoid fever. Thus Landouzy, Picot, Huchard, and others have formulated this conclusion, namely, that the disappearance of the first sound of the heart at the apex or at the base in the course of typhoid fever constitutes an evil sign if the pulse goes as high as 110, and that if the sound be absent and the pulse-rate increases in excess of this number per minute, the prognosis is fatal. Of course, any condition of profound depression in the heart or general strength which can extinguish the first sound is more or less grave, but the association of this disappearance with high pulse-rate they consider a very evil omen. Mongour[1] has recently written a confirmatory paper on this theme.

In still other instances the heart-sounds are like those of a fœtus, the long pause being absent. This is called "embryocardia," and indicates distinct cardiac feebleness.

These circulatory changes have been chiefly discussed by French clinicians. Bernheim[2] has described a variety of typhoid fever that he calls "forme cardiaque," the chief signs of which are a condition of asystole and cardiac feeblenees. Demange[3] has also written on this topic, and Pótain is quoted by Homolle in his

[1] La Presse Médicale, April 21, 1897.

[2] Association pour l'Avancement des Sciences; Congrès de la Rochelle, 1882.

[3] Revue de Médecin, 1885, p. 1025.

article on typhoid fever, in Jaccoud's *Dictionnaire*, as having found a constant decrease of arterial pressure by means of the sphygmomanometer of Basch. This reduction of pressure is an almost constant symptom, as every one knows who has studied the pulse of patients suffering with this disease.

In other cases, which are rare, comparatively speaking, the pulse-rate remains at or below the normal all through the attack. This is without any particular import, and was thought by the older writers, such as Hufeland, Sauvages, and Berndt, to be quite pathognomonic of this disease. Liebermeister states that a good pulse in typhoid fever rarely rises above 110.

If the circulation distinctly fails, congestion of the veins may develope, but the surface of the body instead of becoming cyanotic or congested in appearance, often becomes pallid and relaxed, a profuse sweat often being present, even though the temperature may be as high as 104°.

Over and above these gradual signs of circulatory failure, sudden collapse from hemorrhage or perforation may develop. (See article on alimentary canal.) A sudden diarrhœa or an attack of vomiting may, however, cause a syncopal attack, and a sudden fall of high temperature due to some complicating state may also do so. Liebermeister, though an ardent advocate of the cold bath, says: "Sometimes a condition resembling collapse is seen to follow a cold bath." So far as prognosis is concerned, care should be taken to separate the collapse of defervescence from that due to grave cardiac degeneration. (For circulatory accidents see chapter on the circulatory system in the stage of convalescence.)

Acute endocarditis complicating typhoid fever has been reported by Carbone.[1] The patient was a young women who had the classical symptoms and lesions of typhoid fever, and from whose endocardium typhoid bacilli were obtained. These bacilli were injected intravenously in various animals, producing the same lesion.

Connell[2] has also recorded a case of infectious endocarditis in

[1] Gazette Medica di Torino, No. 23, 1892.

[2] Montreal Medical Journal, August, 1896.

typhoid fever, due to the staphylococcus and involving the mitral and tricuspid valves.

In connection with this subject, it may be proper to call attention to the profound exhaustion and depression, chiefly manifested at the close of severe typhoid fever, having a tendency to cause death from asthenia. This state was far more frequently met with some years ago, when the infection seemed more virulent than it does to-day, and when the treatment was not so well understood. The condition of the patient has been described by Huxham in his *Essay on Fevers*, 1750, p. 78, in the following words:

"Now Nature sinks apace, the extremities grow cold, the nails pale and livid, the pulse may be said to tremble and flutter rather than to beat, the vibrations being so exceedingly weak and quick that they can scarce be distinguished, though sometimes they creep on surpisingly slow, and very frequently intermit. The sick become quite insensible and stupid, scarce affected with the loudest noise or the strongest light, though at the beginning strangely susceptive of the impressions of either. The delirium now ends in a profound coma, and that soon in eternal sleep. The stools, urine, and tears run off involuntarily, and announce a speedy dissolution, as the vast tremblings and twitchings of the nerves and tendons are preludes to a general convulsion, which at once snaps off the thread of life. In one or other of these ways are the sick carried off, after having languished on for fourteen, eighteen, or twenty days, nay, sometimes for much longer."

The Blood in the Developed Stage of Typhoid Fever. In typhoid fever in the first two weeks of the disease we usually find little if any change in the red corpuscles, unless an active diarrhœa be present, in which case there may be concentration of the blood-cells. In the third week the red cells begin to decrease, and may get as low as in cases of pernicious anæmia. The lowest point is reached about the end of the first week of convalescence, when they gradually begin to increase. The hæmoglobin follows the red cells, as might be expected, and the degree of the anæmia is in direct proportion to the severity of the case in most instances. The most noteworthy fact about the blood in this fever is that,

as rule, there is no constant increase in the leucocytes unless some intercurrent inflammation is set up. Cabot asserts, however, that sometimes leucocytosis does occur without any complication that can be found. On the other hand, in patients profoundly asthenic from this disease complications may not cause leucocytosis. As an illustration of the manner in which these accidents may produce blood changes, the following table of Cabot is of interest:

			Leucocytes.
Perforation.	Case I	(*a*). Five days before perforation . .	8,300
		(*b*). At time of perforation . . .	24,000
	Case II.	At time of perforation . . .	18,500
Phlebitis.	Case I	(*a*). Two days before onset . . .	6,400
		(*b*). At time of onset	12,900
		(*c*). One week later	10,100
	Case II	(*a*). One week before onset . . .	4,800
		(*b*). At time of onset	16,200
Otitis media.	Case I	(*a*). At entrance	5,300
		(*b*). Mastoid abscess	16,400
	Case II	(*a*). At entrance	8,400
		(*b*). Two weeks later, after opening drum membrane (sero-purulent discharge)	11,200
	Case III	(*a*). At entrance	7,320
		(*b*). Otitis	14,000

A freely discharging otitis soon ceases to cause leucocytosis—*e. g.*, a case of serous otitis media seven days after puncture, but still discharging freely, showed but 5320 white cells per cubic millimetre.

An abscess of the buttock raised the count from 8000 to 11,200, and a hemorrhage from 8000 to 11,300.

As with all inflammations, it is the increase in the polymorphonuclear cells which is chiefly indicative.

The real value of discovering alterations in the blood in typhoid fever is very great for diagnostic purposes. Increased leucocytosis will give us reason to believe that there is present, and make us search for, some complicating inflammatory focus, such as pneumonia, perforation, cholecystitis, phlebitis, or abscess in any part of the body, as in the liver. Further, it may render a case of suspected typhoid fever clearly one of appendicitis or some other inflammatory affection.

The study of leucocytosis is useless to us in separating malarial fever from typhoid fever, for in neither affection does it occur, and the same statement holds true as to tuberculosis unless the latter is accompanied by coincident infections with pus organisms, when leucocytosis may be present.[1]

The blood in typhoid fever should not be examined after a bath, as this may cause a temporary leucocytosis in the peripheral vessels.

The bacillus of Eberth is very rarely found in the blood, but a recent case of interest has been reported by De Grandmaison and Cartier.[2] They report the case of a woman who was admitted to the hospital suffering from the results of an abortion, who presented typical typhoid symptoms, and whose blood gave the positive Widal reaction, and from whose blood they obtained pure cultures of the bacillus of Eberth.

The Spleen. The changes produced in the spleen are usually developed during the fourth week of the disease. Hoffmann found nine cases of infarction of this organ in 250 autopsies, and seven of these died in the fourth week. Griesinger believed infarction of the spleen to be found in 7 per cent. of fatal cases, and Liebermeister believed these lesions to be responsible for the production of peritonitis in many cases where this condition arises independently of perforation. Sometimes the infarction results in the formation of a large abscess filling the greater part of the organ. Liebermeister records a case in which after death from general peritonitis the spleen, which was three times its natural size, was found transformed into a huge abscess, making seven-eighths of its bulk. No perforation of the abscess wall had occurred.

Under the name spleno-typhoid, Eiselt[3] has described a condition in which, according to his description, the spleen bears the brunt of the affection and the intestinal complications are absent.

[1] Valuable studies of these questions are those of Cabot, from whose book on the blood I have quoted, and those of Thayer, Johns Hopkins Hospital Reports, vol. iv. p. 83. Also Ouskow and Aporti and Radaeli, Eleventh Congress for Medical Science, Rome, March, 1894.

[2] La Presse Médicale, February 1, 1899.

[3] La Semaine Médicale, August 27, 1891.

The spleen may be very much enlarged and there may be a perisplenitis with adhesions. In another form the spleen becomes enormous in size, with effusions into the splenic pulp accompanied by high fever lasting for several weeks, and in the third variety the spleen is not so large, but the fever is a very early symptom. In this type a relapsing fever occurs, but Eiselt asserts that spirilla of Obermeier have not been found in the blood in these cases and that they are truly typhoid, because of the intestinal lesions found in some of the fatal cases in the latter forms of the disease and by reason of the source of infection.

The Genito-urinary Tract in the Well-developed Stage of the Disease. It has already been pointed out in an earlier chapter that acute nephritis may usher in an attack of typhoid fever, but such an occurrence is very uncommon, and the development of a nephritis in the later stages of the disease is almost as rare. In such a case the presence of albumin, casts, blood-cells, and, perhaps, pure blood may make a diagonsis easy.

Curiously enough the amount of blood in the urine in such cases is no guide to their severity, because unless the flow of blood has been sufficiently great to decrease the patient's strength it does not represent the degree of renal involvement. Further, it is to be remembered that in some cases in which there is marked hæmaturia, the autopsy fails to reveal marked renal change, or instead of nephritis an infarction. Such cases have been reported by Homburger and by Duckworth, by Sorel, and by other writers. In cases in which there are tube casts and other signs of acute diffuse nephritis, the prognosis may be grave. Osler reports two cases which died. Amat had ten deaths in twelve cases, while Wagner had five consecutive recoveries.

Hemorrhagic nephritis has been recorded by Stevens[1] in association with uræmic symptoms. Relief came by a profuse hemorrhage from the bowels, and recovery occurred.

A very excellent paper on the important subject of albuminuria in typhoid fever has been published by Hewetson, in which he

[1] University Medical Magazine, May, 1896.

has exhausted the literature. He quotes Guimet as having met with albuminuria in children 21 times in 45 cases, and Mason as having met with it in 60 out of 676 cases, of which 45 recovered and 15 died. At the Johns Hopkins Hospital Hewetson found it in 164 out of 229 cases, but tube casts were found in only 103 of these. He also found that the period in which albumin appeared in the urine, so far as he could tell, was in the first week in 66 per cent. of the cases ; in the second week in 75 per cent. ; in the third week in 41.6 per cent. ; while in the fourth week it occurred in 35 per cent. A very interesting thing in this connection is the fact that in none of these cases were there any objective signs of renal disease, any uræmia, or œdema.

Hanford[1] has also shown that albuminuria may occur in typhoid fever without possessing any grave prognostic import, but the quantity of the albumin is in direct ratio, as a rule, to the gravity of the case. Among patients with large amounts of albumin the mortality is usually very high.

Albuminuria occurred in 31 per cent. of 190 cases in Nuremberg, according to Zinn,[2] and epithelium and hyaline casts in 21 per cent.

The urine in typhoid fever is nearly always decreased in amount in the acute stage, and is usually darker in hue than normal, containing a high percentage of solids. Small amounts of albumin may be in it without indicating nephritis, but if casts are present much albumin is usually found, and the diagnosis of nephritis is justified. About 20 per cent. of all cases of this fever show albuminuria at times, but even if mild nephritis develops the prognosis is not, as a rule, grave. Thus in the Johns Hopkins Hospital albuminuria occurred in 164 out of 229 cases, and tube casts in 103 ; altogether 21 out of these 229 cases had definite nephritis, and 10 had red cells in the urine ; 2 suffered from hemorrhagic nephritis, but only 5 of these cases died, and none of them from the renal difficulty.

[1] London Lancet, April 28, 1889.

[2] Münchener Medicinische Wochenschrift, February 14, 1899.

Rostoski[1] found albumin present in the urine 205 times in 346 cases, or in 59.2 per cent. In 37 of these 205 cases the albuminuria was marked and hyaline and epithelial casts were found, proving the presence of an infectious nephritis.

Rostoski[2] reports two cases of renal typhoid fever as follows:

A patient was admitted with severe headache and bronchitis. The urine contained blood, albumin, and epithelial casts. A few days later the characteristic rash and diarrhœa appeared. Widal's reaction gave a positive result. In this case the nephritis passed into the chronic disease.

A woman, aged twenty-six years, was admitted with urine containing blood and albumin, and subsequently epithelial casts. About three weeks after the commencement of the disease Widal's reaction was obtained, and two days later typhoid bacilli were cultivated from the urine. Five days afterward the patient had severe abdominal pain, with vomiting, and moderate collapse. On the next day the whole of the abdomen was exquisitely tender. A little later an impaired percussion note was made out over the ileo-cæcal region, due, as it was thought, to a localized serous peritonitis. The patient gradually improved, and subsequently made a good recovery. The case was very obscure at first. The presence of an acute nephritis was only recognized thirteen days after the onset of the disease. The diagnosis from tuberculosis, malignant endocarditis, and sepsis was very difficult. It was only when Widal's reaction was found in the fourth week of the disease that the nature of the case became obvious. The temperature was not characteristic, but the spleen was enlarged. The signs of peritonitis appeared about the fiftieth day, shortly after the administration of a clyster; previously there had been no intestinal symptoms. The patient also recovered from this complication. Rostoski

[1] Münchener Medicinische Wochenschrift, February 14, 1899. This is the most recent paper on this topic, and contains references to the literature of the subject. The title of the paper "Zur Kenntniss die Typhus Renalis," refers to nephritis complicating typhoid fever, and not that of the form of onset called "nephro-typhus."

[2] These cases are also to be found in an abstract in the British Medical Journal of April, 1899.

expresses the opinion that in every case of nephritis which might be classed as idiopathic, but which has a high temperature, the urine should be examined for typhoid bacilli, and the blood tested for Widal's reaction.

In 147 cases admitted to the German Hospital of Philadelphia[1] in 1898 from the United States Army, albuminuria was present in 57.1 per cent., and true nephritis in 25.2 per cent.

Late in the disease or in convalescence a transient nephritis may develop, associated with pretibial œdema.

Aside from diffuse nephritis due to enteric fever we find that the kidneys may be the seat of suppurative processes, developing, as a rule, in the form of multiple or miliary abscesses. These abscesses are due usually to infection of the organ by the ordinary pyogenic cocci and rarely to infection by the bacillus of Eberth. The latter condition has, however, been recorded by Flexner, who has studied two cases of focal abscesses in the kidney, and found by careful differentiation that this bacillus was the sole cause of the lesion. The urine in these cases was albuminous and contained blood-cells, and at times casts covered with leucocytes. There are few clinical symptoms which can be used to diagnosticate such lesions other than those shown by the urine.

Pyuria arises in typhoid fever either from the kidneys (very rarely) or from the bladder. It varies in severity from the presence of a few pus cells, which are found with difficulty by the microscope, to marked pyuria with quantities of pus. The best study of this subject is probably that of Blumer.[2] He found no less than 16 cases in 60 typhoid fever patients, or nearly 17 per cent. In some the pus was found present when the patient came under observation; in 4 cases it appeared between the tenth and fifteenth day; in 3 between the twenty-second and twenty-eighth day, and in 1 on the forty-second day. Its duration varied from a few days to three months. In nearly all his cases the pus was present in full amount. In some it gradually increased; in others it came in large amount at once. The organisms found in the

[1] Philadelphia Medical Journal, February 25, 1899.

[2] Johns Hopkins Hospital Reports, 1895, vol. v.

urine were the colon bacillus, the typhoid bacillus, staphylococcus albus, and an unidentified coccus. The colon bacillus was found in seven cases, the typhoid bacillus twice, and the staphylococcus once. These observations are important, because it has been said by Karlinski, of Krakow, that he has found the Eberth bacillus in no less than 50 per cent. of all cases. In all probability the differentiation between the colon bacillus and that of the typhoid was not properly carried out.

No case of pyelitis due to the bacillus of Eberth alone has as yet been reported, which is interesting in view of the well-known fact that this bacillus has frequently been found in the kidney after death, and is always found in the renal lymphomata of this disease. Konjajeff[1] asserts that the discovery of this bacillus in the urine indicates the development of these formations in the kidney; but this is improbable, since post-typhoidal pyelitis, not due to this organism, of a membranous type may develop and be associated with a membranous cystitis.

Richardson has recently shown[2] that typhoid bacilli were present in the urine of nine out of twenty-eight cases of typhoid fever; that they were always in large numbers and in practically pure cultures, and that they appear in the later stages of the disease and persist in most cases far into convalescence. Their presence is nearly always associated with albuminuria and casts.

In a still later report Richardson[3] reports sixty-six further cases, of which fourteen showed the presence of bacilli in the urine.

Petruschky[4] has estimated that in one case a single cubic centimetre of urine contained 170,000,000 typhoid bacilli.

Horton Smith[5] examined the urine of seven typhoid patients, with three positive results, and he remarks that the micro-organisms may be so numerous as to cause distinct turbidity of the urine.

[1] Centralblatt für Bakteriologie, 1889.
[2] Journal of Experimental Medicine, 1898, vol. iii.
[3] Journal of Experimental Medicine, 1899, vol. iv.
[4] Centralblatt für Bakteriologie, 1898, xxiii.
[5] Transactions of Medical aud Surgical Society, London, 1897.

Petruschky[1] has pointed out that the bacillus of typhoid is often found in the urine some weeks after the temperature is normal.

To sum up the evidence from a clinical point of view, we find that pyuria in typhoid fever is not a grave sign, but that if the specific bacillus is found in the urine the patient must be kept under observation till it disappears, since it may lead to serious mischief.

Pyonephrosis has been recorded by Fernel.[2] The patient, who had previous to typhoid fever suffered from intermittent hydronephrosis, developed a fluctuating abdominal tumor, which proved to be a pyonephrosis containing a pure culture of the bacillus of Eberth.

A case of typhoid cystitis has been recorded by Houston.[4] A woman, aged thirty-five years, had suffered from cystitis for a long period of time; the urine was strongly acid, turbid, contained a small quantity of albumin as well as squamous epithelium, leucocytes, and some bacteria. A bacillus with all the characteristics of that of typhoid was cultivated, and her blood gave a marked typhoid reaction of 1.01. A second examination of her urine produced similar results; although the patient was kept in the hospital for six weeks, there were no other typhoid symptoms and no febrile movement.

In all probability this is a case in which the disease had been so mild at some previous time as not to attract attention, but the bladder-infection had persisted.

Profuse urinary flow is sometimes seen in the latter part of defervescence and in convalescence. It may amount to ninety ounces in twenty-four hours for many days. This has usually no great significance. Hutchinson[3] has reported a case of diabetes mellitus following typhoid fever.

The Alimentary Canal in the Developed Stage. Reference has already been made to pharyngeal typhoid lesions in the stage of onset. A more or less severe inflammation of the

[1] Centralblatt für Bakteriologie, 1892, xiv.

[2] Gazette des Hôpitaux, 1897, No. 10.

[3] Transactions of Association of American Physicians, 1888, vol. iii.

[4] British Medical Journal, January 14, 1898.

pharynx is to be found in nearly all severe cases of typhoid fever if it is sought for, and it is sometimes sufficiently marked to cause the patient to complain of his throat. As a rule, the lesions consist in congestion of the mucous membrane with swelling of the glands in this part of a character similar to that met with in other parts of the alimentary canal. Pharyngeal symptoms may develop in convalescence (which see); sometimes membranous pharyngitis coming on in the third week may cause death, and Taupin[1] records a case in which it asserted itself in a case of typhoid fever complicated with measles.

Gerloczy,[2] a physician of Budapest, has recorded a case of a girl of fourteen years, who suffered from typical typhoid fever with swelling of the submaxillary glands and the development of a membrane in the pharynx. The case had pulmonary œdema, and membranous pharyngitis, laryngitis, and bronchitis.

Not only are inflammatory changes found in the pharynx in this stage of typhoid fever but also in the œsophagus, where, of course, they are apt to be more moderate than in the pharynx because of the lack of lymphoid tissue. Usually swelling of the glands in the mucous membrane is to be found on inspection. As the disease progresses these changes may become ulcerative and severe. Louis and Jenner have seen cases of typhoid ulceration of the œsophagus, and that Roderer and Wagner have seen œsophagitis, as have also Eichhorst and Reimer, and again, Chauffer and Cornil have described a condition of infiltration of the mucous membrane of the œsophagus with a formation of miliary abscess. These changes will be found discussed in the chapter dealing with the stage of convalescence.

Symptoms peculiar to the *stomach* are comparatively rarely met with in typhoid fever, unless dietetic errors have caused them, or unless by the excessive use of drugs or stimulants its functions become perverted. On the other hand, when gastric symptoms arise, either as the result of the causes just named, or

[1] Journal des Connaissances Méd. Chirurgicale, 1839.

[2] Deutsche med. Wochenschrift, April 14, 1893.

because of some unusual feature of the disease, they are apt to be not only annoying but difficult of control. Aside from moderate gastric catarrh due to the fever and associated with a condition of insufficient and inefficient gastric juice, which is peculiarly marked in these cases, the unusual symptoms vary from hiccough, which is really an affection of the diaphragm produced by a reflex from the stomach in many cases, to vomiting, and from discomfort in the epigastrium to severe pain. Disregarding the moderate form of hiccough seen so often accompanying ordinary indigestion, we now and again meet with cases in which this symptom becomes not only annoying but exceedingly dangerous, in that it causes rapid exhaustion and failure of the heart, apparently by some associated vagal neurosis, over and above the great drain upon the patient's strength. Numerous cases are on record in which this complication has resulted in great danger or even in death.

Vomiting in typhoid fever may be an unimportant or very grave complication. Often it occurs because of indigestion or irritability of the stomach, and stops as soon as the diet is altered or the quality and mode of using stimulants is changed. Its gravity depends largely upon its persistency, because if it ensues on taking food the patient speedily dies from lack of nourishment, and if it is of the incessant type, resembling the status epilepticus in its constancy and spasmodic character, the patient retching incessantly whether the stomach is empty or not, death is imminent because of direct exhaustion. Such cases are not common, but when they occur the prognosis must be very grave. Sometimes it would seem as if the vomiting was caused by a neurosis or by poisoning of the vomiting centre in the medulla.

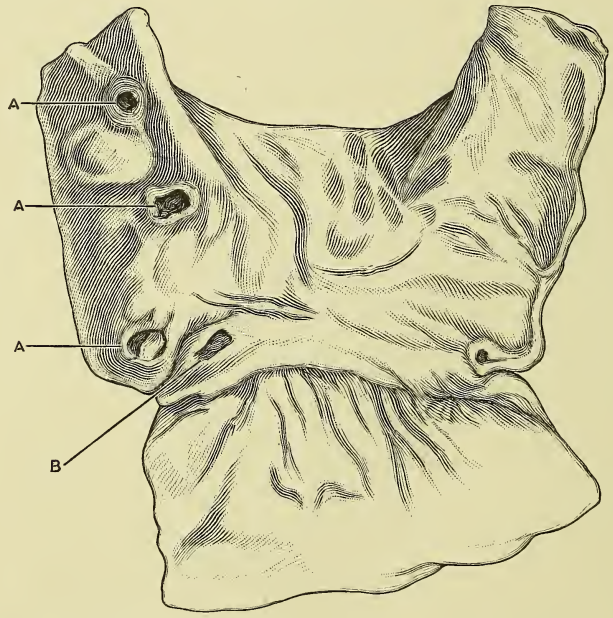
Still more rarely in typhoid fever the vomiting arises from ulcer of the stomach.

Hemorrhage from the stomach is very rare in typhoid fever and is almost unknown. Pepper states that typical typhoid ulcers may be found in the stomach, and from them it is possible that hemorrhage may occur. Soltau Fenwick[1] has recorded a case in which

[1] Disorders of Digestion in Infancy and Childhood, 1897, p. 386.

typhoid ulcers nearly perforated, and another in which they did perforate but general peritonitis was prevented by the liver becoming adherent to the wall of the stomach. Death occurred in this case from profuse hemorrhage from one of these ulcers. I have only met with one case in which hæmatemesis took place. A woman of twenty-eight years, who was seized with a very severe attack of the disease died at the end of the first week immediately after vomiting a large amount of blood and passing a great

FIG. 15.



Drawing of the pyloric end of the stomach in a case of enteric fever. *a*, acute perforating ulcers with clean bases; *b*, an ulcer with adherent slough. (W. SOLTAU FENWICK.)

quantity by the bowel. No autopsy was held, and in all probability the blood had entered the stomach from the small bowel. The following cases are those of Fenwick's:

"Fig. 15 represents a drawing of a stomach taken from a girl, eight years of age, who succumbed during the third week of enteric fever. Four well-defined ulcers were found in the pyloric

region, one of which presented a loosely adherent slough. The edges of the ulcers were sharply defined and somewhat undermined, while their bases were situated in the submucous and muscular coats of the organ. On microscopic examination the lymphoid tissue of the stomach was found to be enormously increased, and the supposition that the ulcers originated in disease of the solitary glands was confirmed by the appearance of the smallest one. From these facts it would appear that under certain circumstances disease of the solitary gastric glands may give rise to a form of perforating ulcer of the stomach which closely resembles the idiopathic type of the disease.

"A girl, thirteen years old, was admitted into the hospital with the symptoms of typhoid fever of eight days' duration. Vomiting occurred once or twice, but there was no complaint of epigastric pain. At the end of the fourth week of the disease, when the temperature had begun to decline, the patient was suddenly seized with severe hæmatemesis, after which she became unconscious and died. At the necropsy the anterior wall of the stomach was found to be adherent to the under surface of the liver. Scattered over the inner surface of the stomach there were numerous sharply defined ulcers, the largest of which was about the size of a florin. The edges were thin and undermined and the base was formed by the muscular or peritoneal coat. In the first part of the duodenum there was an ulcer of a similar character, while the whole of the intestine, from the jejunum to the rectum, was riddled with typical typhoid ulcers."

Osler has reported the following cases to Keen:

"John M., aged forty years, was admitted August 21, 1890, with a history of illness of some weeks' duration. The chief symptoms were headache and fever. The blood examination was negative. There was a very definite rose-colored eruption. The temperature was never high, not rising above 103°. On the 27th he vomited, and in one of the attacks he brought up a dark greenish-brown fluid containing red blood-corpuscles in a condition of disintegration, and a clot of blood about 3 by 2 cm. in diameter. On the 29th, 30th, and 31st the stools were very dark in color,

and evidently contained blood, and several times he vomited very dark material. He became very anæmic, but made a good recovery.

"Alberta C., colored, aged twenty years, admitted June 14, 1894. This patient was admitted in the third week of the disease. On that afternoon she had had a hemorrhage from the bowels. She was bleeding quite freely on admission. Between 6 and 8 P.M. she had five large stools of almost pure blood with clots. Throughout the following day she was extremely feeble; temperature was normal; patient was delirious. On June 16th there was no further bleeding from the bowels. Toward evening the patient was delirious, and her condition was very bad. At 8.15 P.M. she vomited 100 c.c. of dark bloody fluid, which contained blood coloring-matter and red blood-corpuscles. She sank, and died that evening.

"Dr. H., aged twenty-two years, admitted January 9, 1896. He had a very severe attack, with persistent fever, which resisted the baths. These, though given from the outset, did not check the onset of quite active delirium. On January 25th, about the eighteenth day of the disease, the abdomen was a good deal distended; there was moderate diarrhœa and less delirium. He seemed to be doing very well. He had had no special gastric symptoms. In the afternoon he quite suddenly sprang up in bed and vomited a quantity of dark blood. The amount was difficult to estimate, as it went all over the bed linen. Part of it was collected, and Dr. Parsons estimated the amount to be about 200 c.c. It contained much débris and red blood-corpuscles. The staining on the sheets was quite red. On the 26th the temperature was between 103° and 104°, and in the afternoon at 3.05 he vomited between 200 and 300 c.c. of almost pure, bright-red blood. The pulse became more rapid, but these two hemorrhages did not appear to have any injurious influence. His temperature gradually fell and was normal on the 31st. He made an uninterrupted recovery after a most severe attack."

Weiss[1] records a case of a soldier, aged twenty-two years, who

[1] Wiener Med. Presse, 1888.

died from profuse gastric hemorrhage about the beginning of the third week of typhoid fever. This was preceded by intestinal hemorrhage. As no statement is made as to whether a post-mortem confirmed the diagnosis, the case is to be considered as a doubtful one.

One of the first facts which attracts our attention in regard to the *intestine* during typhoid fever is that many cases of this disease are recorded in which at the autopsy no signs of typhoid fever could be found in the intestines. Some of these have not been as carefully studied as they should be, but others are certainly authentic. Thus Du Cazal[1] has recorded two instances in which the closest inspection failed to show intestinal lesions, yet typhoid bacilli, which responded to all tests, were found in the spleen, and the symptoms of the disease were present in life. The spleen, mesenteric glands, and kidneys were swollen and congested. Bacilli of typhoid fever were obtained not only from an abscess in the spleen, but also from vegetations in the mitral valves and from a hemorrhagic plaque on the surface of the brain. Banti[2] and Karlinski[3] have reported similar cases not so well proven. Karlinski's cases numbered three.

Nichols and Keenan[4] have reported nine cases of typhoid fever without intestinal lesions. So, too, Flexner and Harris[5] have recorded such a case, and Chiari and Kraus met with seven out of nineteen cases in five months.

Goodall[6] reports two cases of enteric fever, fatal during the third and fifth week respectively, in which there was no intestinal ulceration. The first patient was a boy of thirteen years, who had been ill a fortnight when admitted to the hospital; the second was a man of thirty years, who had already been ill ten days. Both of them showed all the clinical evidences of typhoid fever,

[1] Bulletin et Soc. Mem. Méd. des Hôp., 1893, p. 243, and Le Bulletin Médical, April 16, 1894.

[2] Archiv. Italiennes de Biol., December, 1887.

[3] Wiener Med. Wochenschrift, 1891, pp. 470 and 511, and 1897, vol. ii. p. 1850.

[4] Montreal Medical Journal, 1898, xxvii. p. 9.

[5] Johns Hopkins Hospital Bulletin, 1897, viii. p. 259.

[6] Clinical Society's Transactions, vol. xxx., 1897.

and in each there was a swelling of Peyer's patches without ulceration. Similarly Fagge[1] records the case of a man of thirty-three years, who had typhoid fever, and whose only lesion in the intestine consisted of one ill-defined purplish-red patch about the size of a shilling, situated a foot above the valve and a little higher up; another patch with a brush surface, which was visible only when it was examined under water. So, too, in November, 1880, Moore showed before the Pathological Society of Dublin a case of enteric fever in which there was no disease of the glands of the ileum, while the spleen was extremely large, soft, and friable, and Peyer's patches were noted appearing less distinct than usual, though with no hyperæmia, and did not present the shaven-beard appearance. Sydney Phillips has reported to the Clinical Society, 1891, two cases, fatal after the third week, with no ulceration. Goodall points out that out of sixty-three autopsies he has held in cases of enteric fever at the Eastern Hospital he has met with absence of ulceration in five cases; in two of these death took place early, on the eighth and tenth days; in two others, as the result of some complication, on the thirty-second and seventy-third days.

Other cases have been recorded by Beatty,[2] Church, and Coupland.

Again, Hodenpyle,[3] of New York, has contributed a paper upon this subject, reporting a case of undoubted typhoid fever in which the intestinal lesions were absent. Brunschwig[4] has also recorded a case of this kind, and Hoeffel[5] has done likewise, there being in his case but slight swelling and reddening of a few Peyer's patches. Schultz claimed to have met with twenty-one cases out of 300 autopsies of this disease without the characteristic ulcers in the ileum; but there is doubt as to the correctness of his statement.

[1] Pathological Society's Transactions for 1876.

[2] British Medical Journal, June 16, 1897, p. 148.

[3] British Medical Journal, December 25, 1897.

[4] "Is the Lesion of Peyer's Patches a Constant Symptom of Typhoid Fever?" Strasburg Thesis for 1870.

[5] Gazette Médicale de Strassburg, 1871, No. 14, p. 167.

Since the above remarks and quotations were put in type a paper upon this subject has been published by J. H. Bryant.[1] In it he reports the case of a child of twenty-one months, who died of typhoid fever at the end of the third week, and whose blood before death gave the Widal test. The autopsy showed that the heart weighed one and one-half ounces, and appeared to be normal. The arteries, mouth, pharynx, œsophagus, and stomach were normal in appearance. The ileum also appeared to be normal. There was no ulceration, and the Peyer's patches were not swollen or discolored. Nowhere in the intestine could any sign of recent typhoid ulceration be found, and there was not any appearance suggesting a healing or healed typhoid ulcer. The peritoneum was normal. The liver weighed sixteen ounces, and had a normal appearance. The gall-bladder and pancreas were normal. The mesenteric glands were much enlarged, and felt very soft; on section they presented a pinkish-gray color, and appeared to be in a condition of acute inflammation; there was no sign of suppuration or caseation in any of them. The suprarenal capsules were normal. The kidneys weighed three ounces; they were pale. The spleen was a little enlarged.

That the case was one of true typhoid fever is proved by the results of careful bacteriological study of the tissues. As Bryant well says:

"Nothing unusual was anticipated before the necropsy took place. It was expected that the usual typical ulceration of the Peyer's patches of the lower part of the ileum would be found, and great surprise was expressed when no swelling, discoloration, ulceration, or other abnormalities whatsoever could be detected in the Peyer's patches, solitary glands, or mucous membrane of any part of the intestine. I thought at first an erroneous diagnosis had been made, and suggested that the symptoms might have been accounted for by the broncho-pneumonia which was found, although the character of the pyrexia was against this view. After finding the enlarged mesenteric glands, I suggested that, after all, it

[1] British Medical Journal, April 1, 1899.

was most probably an anomalous case of typhoid fever without any lesion of the intestinal mucous membrane. Cultures from the enlarged mesenteric glands yielded an almost pure culture of the bacillus typhi abdominalis. The slight clotting of the milk inoculated from the first broth culture taken directly from the glands was probably due to a slight contamination with the bacillus coli communis. It will be noticed that coagulation did not take place until after forty-eight hours, and then it was only slight. I could not find any colonies of the bacillus coli communis on the gelatin plates, although I looked and carefully examined for them, so that if present originally the number must have been insignificant. The bacillus obtained from the gelatin plates gave the characteristic positive and negative reactions of the bacillus typhi abdominalis, namely, did not produce gas in any media, did not cause milk to clot, did not produce indol, did not produce acid, did not liquefy gelatin, and further, these bacilli obtained from a recent culture and treated with both 50 per cent. and 5 per cent. serum from a typhoid patient, and also from an immunized rabbit, clumped together in a manner characteristic of the bacillus typhi abdominalis."

Byrant also quotes the following cases not mentioned in my text before I read his article:

Thue,[1] in 1889, described a case in which during life the fever was of a recurrent type, and the spleen was found to be considerably enlarged. At the necropsy slight swelling only of Peyer's patches was found. The bacillus typhi abdominalis is stated to have been obtained from the spleen and kidneys, but is not sufficiently identified as such.

Vaillard,[2] in 1890, reported the case of a young soldier who died after an illness of three days' duration. The chief symptoms were headache, epistaxis, pyrexia, constipation, retraction of the neck, and coma. At the necropsy congestion of the lungs and meninges was found, but there was no intestinal lesion. The

[1] Jahresbericht über die Fortschritte (Baumgarten) 1889, 196.

[2] La Semaine Médicale, March, 1890, p. 94.

bacillus typhi abdominalis was obtained by culture from the spleen, lungs, and spinal cord; streptococci were also obtained from the spleen and meninges.

Guarnieri,[1] in 1892, described a case of typhoid fever which during life presented the characteristic symptoms of the disease. No intestinal lesion, however, was found at the necropsy, but the bacillus typhi abdominalis was obtained by culture from the biliary passages, liver, and spleen.

Vincent,[2] in 1893 described the case of a man, aged thirty-five years, who died about the twelfth day after the onset of a severe illness characterized by pyrexia, diarrhœa, purpura, and coma. At the necropsy the Peyer's patches were found to be normal; the mucous membrane of the intestine, however, was congested. The spleen weighed 230 grammes; the mesenteric glands were not enlarged; bilateral pulmonary congestion was found. The bacillus typhi abdominalis and streptococci were obtained from the spleen, liver, kidneys, and heart.

Osler mentions a case. The patient was a man, aged sixty years, who was admitted into the hospital under his care. He had been ill for about two months, and on admission was found to be suffering from shortness of breath, and presented signs of pneumonia affecting the lower lobe of the right lung. Death took place twenty-four hours after admission. A diagnosis of senile pneumonia was made during life. At the necropsy the lower lobe of the right lung showed fresh pneumonia passing on to a condition of gangrene. There was no intestinal lesion. The organs were submitted to a bacteriological examination by Flexner, and pure cultures of the bacillus typhi abdominalis were obtained from the lungs and spleen.

Mettenheimer[3] records an epidemic of typhoid fever occurring in the army in which in twenty-one cases the intestinal lesions

[1] Rivista Générale Italiana di Clinica Medica, 1897; Baumgarten's Jahresbericht, 1897, 234.

[2] Annales de l'Institut Pasteur, February, 1893.

[3] Jahresberichte über die Gesammte Med., 1872, Bd. 2, p. 235.

were entirely limited to the colon. Banti[1] and Karlinski[2] have also reported cases of this character.

A case is recorded in Cheadle's[3] service at St. Mary's Hospital of a child of three years who died of typhoid fever, and at the necropsy no ulceration was present in the intestine and Peyer's patches appeared to be normal. Beatty[4] records two cases with a similar condition present.

Diarrhœa is speedily ceasing to be a fairly constant symptom of the disease. As a matter of fact, it is in a very large proportion of cases supplanted by constipation from the beginning to the end of the malady, although classical works nearly all regard looseness of the bowels, amounting to three or four stools a day, as the usual condition in average attacks. This is particularly the case in the typhoid fever of children, in whom constipation occurs even more commonly than in adults.

Students very often seem to have the idea that the absence of diarrhœa in a given case is an important point against the diagnosis of typhoid fever. On the contrary, it is so often absent that its absence is of no negative value whatever, although its presence possesses more importance. Certainly constipation is much the more frequent state as we meet the disease in Philadelphia, and as Osler well points out, diarrhœa occurs in Baltimore in not more than 30 per cent. of his cases, and is an active form in only about 12 per cent. So, too, we find that in Curschmann's[5] clinic from 1880 to 1892, diarrhœa was met with in only 25 per cent. of the cases (1626 cases). Phillips tells us that of 200 consecutive cases in St. Mary's Hospital, London, diarrhœa occurred in 115, constipation in 48, but in many of these cases diarrhœa had been set up by a purge given before the diagnosis was made, so that his experience in no way militates against the statistics just cited.

[1] La Riforma Médica, 1887, p. 1448.
[2] Wiener Med. Wochen., 1891, pp. 470 and 511.
[3] The Lancet, July 31, 1897, p. 254.
[4] British Medical Journal, January 16, 1897.
[5] Deutsche Archiv. f. klin. Medicin, 1895.

In the Maidstone[1] epidemic 50 per cent of the cases were constipated. Murchison found it in 93 out of 100 cases.

When the diarrhœa is excessive, amounting to ten and twenty stools a day, the diet has usually been faulty in the extreme, or ulceration of the large bowel, amounting to a dysenteric state, is generally present. The character of the stools is usually, in the cases with moderate diarrhœa, quite typical, but green stools in typhoid fever are occasionally met with. They have been referred to by Dreschfeld in Allbutt's *System of Medicine*, the discoloration being seen during convalescence. Quill[2] has recorded a case in which bright-green material was vomited on the eighth day, and later the patient passed bright-green fluid stools. There was great pain in the back. Garrod, Drysdale, and Kanthack[3] report three cases. The stools resembled chopped parsley, and the liquid portion of the stools when filtered off contained biliverdin, which was probably responsible for the discoloration of the excreta.

The next point to be considered in this connection is whether diarrhœa is a sign of mild or severe infection. The consensus of opinion seems to be that diarrhœa is usually more active in serious cases. Whether this is an instance of "purging as an effort at elimination," a favorite theory with those who are fond of using purgatives and so-called intestinal antiseptics, with the idea that by so doing they eliminate poisons and prevent their formation, or whether it is a manifestation of severe ulceration of the bowel with an associated catarrh, is difficult to determine. Ord[4] agrees with the view that diarrhœa is usually associated with ulceration, and his opinion has been confirmed by the autopsies he has seen. Peabody states the case exactly opposite to this view. That Ord's view is not correct seems proved by the fact that advanced ulceration is often found in cases which have not had diarrhœa and cases of marked diarrhœa are seen in which the

[1] Poole. Guy's Hospital Reports, 1898. Wrongly labelled on cover, 1896.

[2] British Medical Journal, October 22, 1898, p. 1252.

[3] St. Bartholomew's Hospital Reports, vol. xxxiii.

[4] Transactions Association of American Physicians, 1888, vol. iii.

autopsy does not reveal much intestinal ulceration. In Bryant's case, already quoted, diarrhœa was active, yet no intestinal lesions were found. In all probability diarrhœa is neither indicative of a severe nor a light attack in many cases, although if it be violent the exhaustion produced by the discharges may seriously imperil the patient's chances of recovery. This view is strongly advocated by Sydney Phillips, who regards diarrhœa as a symptom adding danger to the progress of the typhoid, as he believes it prevents absorption of nutriment and drains the body of fluid; he is therefore distinctly opposed to the so-called "purgative treatment."

Closely allied to this question of diarrhœa and constipation is that of the gravity of tympanites, a condition almost always present at some time during the course of even the mildest attacks, and, as a rule, less frequently present in cases with active diarrhœa than in those with constipation, although a great accumulation of gas in the intestines is also met with in some instances in which the bowels are moving quite frequently. As a rule, such passages are small in bulk and unsatisfactory in quantity, and are usually quite fetid. The gravity of tympanites as a symptom depends chiefly upon its ability to do harm, and this harm is in direct proportion to the degree of its interference by pressure with the functions of the thoracic and abdominal organs. That is the strain put, by the distention, upon those parts of the bowel-wall which are weakened by ulceration and in danger of perforation from this cause, or to the stretching of the floor of an ulcer, thereby inducing hemorrhage. The degree of tympanites is not always a definite guide as to the damage it may do. It may be extreme in one case and moderate in another, and yet in the first instance very little harm seems to be done by it, while in the second instance, either by reason of cardiac susceptibility or peculiar application of the pressure, the injury may be grave. While, therefore, the degree of the tympanites is in direct ratio to its evil effects, as a rule, cases are continually met with in which it is excessive and yet in which no bad results ensue. When the tympanites is very excessive constipation may result from paralytic distention of the gut, and, on the other hand, the

paralysis or relaxation of the bowel may, by preventing peristalsis, permit the accumulation of gas.

Pain in the abdomen is very distinctly a symptom of the early stages of the disease, and in many cases is due to gas produced by fermentation. The pain is usually wandering and is not constantly in one spot, and if it becomes fixed it probably depends upon a localized complication. Pressure upon the belly-wall is apt to increase the pain. It is, however, a noteworthy fact that later on in the disease, when tympanites is often excessive and the bowel greatly distended, there is apt to be little or no pain even on pressure, perhaps because the atony of the muscular coat of the bowel prevents griping, and the tenderness of the first stage of swelling and inflammation is supplanted by a state of local and general nervous torpor.

Hemorrhages. The frequency with which hemorrhages occur varies greatly in different epidemics, independently of any specific line of treatment over and above rest in bed. Lack of such rest at any stage of the malady certainly predisposes the patient to this accident.

In 861 cases of this disease without the cold bath in Liebermeister's clinic at Basel, hemorrhages occurred 72 times, or 8.4 per cent. Griesinger met with 32 cases in 600, or in 5.3 per cent.; and Louis found them in 5.9 per cent., excluding mild cases; Berg, in 1626 cases, met with them in 5.5 per cent. The younger Wunderlich has recorded 98 cases of typhoid fever without the bath, with hemorrhage in 2 cases, or about 2 per cent. Kraft[1] found in the study of intestinal hemorrhage in typhoid fever that it occurred in 4.24 per cent. of cases, and, curiously enough, that women were more frequently attacked than men, while on the other hand, more males died from this accident than females. He does not think that the prognosis depends directly upon the amount of blood lost. We find, therefore, that in 1559 cases treated without the cold bath there were 99 hemorrhagic cases, or 5.2 per cent.

[1] Centralblatt f. die med. Wissenschaften, 1893, p. 137.

On the other hand, we find that in bathed patents Wunderlich, Jr., records 155 cases with 16 hemorrhagic patients, or 10.3 per cent. Immermann, at Basel, records 146 cases with 6 hemorrhages, or 4.1 per cent.; and Liebermeister, 882 cases with 45 hemorrhages—1183 cases, or 6.8 per cent.

This is shown best by the following table:

WITHOUT BATH.

	Cases.	Hemorrhages.	Per cent.
Liebermeister	861	72	8.4
Griesinger	600	32	5.3
Wunderlich, Jr.	98	2	2.0
Total	1559	106	5.2

WITH BATH.

	Cases.	Hemorrhages.	Per cent.
Liebermeister	882	55	6.2
Immermann	146	6	4.1
Wunderlich, Jr.	155	16	10.3
Total	1183	77	6.8

To these may be added: In America, with baths, Wilson's 140 cases with 10 hemorrhages, or 7 per cent.; Osler's 356 cases with 12 hemorrhages, or 3.4 per cent.[1]

It is interesting to note in this connection that Fitz places the general frequency at 5 per cent. and Loomis at 5 per cent. It is, however, only fair to state that Goltdammer, from nearly 20,000 cases, concludes that the bath does not increase hemorrhages. Brand claims that they are less frequent in the bath treatment, as do also Tripier and Bouveret; but Roland G. Curtin tells us that upon investigation he found that since the cold-water treatment has been instituted the number of hemorrhagic cases has considerably increased, according to the hospital records that furnish the data, and in addition the mortality of the hemorrhagic cases is largely increased, viz., from five in seventeen, less than one-half, to twenty-five in forty-three cases, or over one-half; and, further, on inquiry he found that in two of his tabulated cases the hemor-

[1] Only 299 were bathed.

rhage seemingly took place while the patient was in a bath, and in one case immediately after a bath.

An important point in this connection is the question as to the real danger to the patient from hemorrhage. In this opinions greatly differ. Thus Fitz tells us that it is always a serious symptom, but rarely fatal in private life; but that it may be very disastrous is shown by the fact that Liebermeister mentions 49 deaths due to this cause out of 127 deaths; Murchison, 53 deaths from hemorrhage out of 100 deaths, and Homolle, 44 per cent. in 498 deaths. Osler asserts that death occurs in from 35 to 50 per cent. of hemorrhagic cases. Out of Griesinger's 32 cases 10 died, 7 of these within four days of the hemorrhage. Liebermeister tells us that among his own cases, 38.6 per cent. died when they had hemorrhage, as against 11 per cent. without this accident, and Tyson tells us that the 7 per cent. mortality in his cases under the bath treatment was due entirely to hemorrhage or perforation. It is evident that Osler's percentage is about correct.

On the other hand, it has been noted by some clinicians that if the hemorrhages are not sufficient to produce profound exhaustion the patient often does better after their occurrence than before. This fact was at one time insisted upon by Dr. Alfred Stillé, and it is certainly true in a certain proportion of cases.

While, as a general rule, the danger is in direct ratio to the quantity of blood lost, recovery may occur even after enormous quantities have been passed. I have had a case which recovered in which no less than four pints of blood were lost at one bleeding and Phillips and Wakefield, in 1882, saw a patient who bled "two chamberfuls" and recovered.

As a rule, bleeding from the bowel in typhoid fever arises from ulceration of an arterial twig, but cases do occur where blood comes from a vein which has been opened by ulceration. Phillips has recorded such an instance.

In children hemorrhages from the bowel are more rare than in adults because the intestinal lesions are not so marked, as a rule.

As an illustration of how rarely intestinal hemorrhage complicates typhoid fever in children, the statement of Simon, that in

twenty-one years of practice he had encountered only three cases, is of interest.

Hillier, on the other hand, met with hemorrhage in 4 out of 30 cases. The younger the child the less is the liability to this accident.

Perforations. Perforation of the bowel in typhoid fever bears no relation to the severity of the general symptoms. In many cases the reporting physician states that the attack of enteric fever was mild, so that in 444 cases collected by Fitz, fully 200 were of this class. In fourteen of the cases the patients belonged to the class known as "walking typhoid" cases. Thus Bennett[1] reports the case of a man who, because of cardiac dropsy, was admitted to St. Thomas' Hospital. He was purged and allowed to eat heartily. Two weeks later he began to suffer from abdominal pain, and the next day death took place from perforation due to typhoid fever. No typhoid symptoms had been present. Finncane[2] reports a case of a man apparently well till two days before death, when typhoid perforations occurred, and Kleinwachter[3] speaks of a woman who, till forty-eight hours before her death, was at business and who was suddenly stricken and died from this cause.

When perforation occurs the symptoms are apt to be ushered in by agonizing pain, usually felt in the appendicular region, which may be severe enough to rouse the patient from a considerable degree of coma. The belly wall speedily becomes tense and tympanitic, and all the symptoms of a general diffuse peritonitis speedily ensue. The pain may, however, not be persistent, but pass away or become modified, as the peritoneal inflammation resulting from the escape of fecal matter into its cavity becomes more and more septic. The pulse becomes rapid and running, and collapse may speedily assert itself. When this occurs death speedily comes on, the patient dying in a few hours, or, again, he may rally and survive for several days. Early death is, how-

[1] Transactions of the Pathological Society, London, 1866, xvii. 121.

[2] Lancet, 1889, ii. 793.

[3] Wiener Med. Press, 1880, xxi. 337.

ever, the more common result. Thus in the collection of thirty-four cases made by Fitz,[1] of Boston, 37.3 per cent. died on the first day, 29.5 per cent. on the second, and 83.4 per cent. in the first week. During the second week nine died, in the third week four died, and two other cases lived thirty and thirty-eight days respectively.

If collapse does not ensue the rally of the system results in a rise of the temperature to a point higher than before the accident, and this movement is often accompanied by chills and rigors. Usually by the second or third day the peritoneal symptoms become more and more marked, the condition of the patient more and more asthenic and depressed, and death results by the fourth day from a general peritonitis with toxæmia from the absorption of toxic materials. In other cases the onset of the perforation is insidious, the belly before the perforation may have been moderately tympanitic, but now becomes intensely hard and swollen; the pain, which in some cases is so severe, does not develop, but the great fall in fever followed by a rise, and this again by rigors, it may be, give evidence of the grave accident which has occurred. The pulse becomes increasingly rapid and running, and the respirations more and more costal and less and less diaphragmatic, until the patient sinks out of life, without much, if any, suffering, in much the same manner as one sees death come to a case of diffuse septic peritonitis due to a pus-tube or an old appendicitis. In such cases the perforation is usually very small, and is so surrounded by adhesions that the escape of the intestinal contents is very gradual and insidious, infecting the peritoneum without the escaping fluid being copious enough to produce great pain or infection. This possibility of perforation of the bowel taking place insidiously has been mentioned by Sydney Phillips,[2] of London. To use his words: "In some cases of typhoid fever where nerve-tone is already lost and the tympanitic belly is soft and doughy, perforation and after-peritonitis

[1] Transactions of the Association of American Physicians, 1891, vol. vi.

[2] British Medical Journal, November 12, 1898.

may occur almost insidiously with little pain, collapse signs or alterations in temperature." The former type of case is illustrated by the case of a medical student under my care, who while convalescing from a very mild attack of the disease, and who had had a normal temperature for several days, was seized at midnight with agonizing pain in the epigastrium, so severe that he implored his father to relieve him or kill him in order to stop his suffering. He rapidly passed into collapse, and died in eight hours.

The insidious form is shown by the case of a man who came under my care in the third week of the disease, much exhausted and emaciated, but without very high fever at any time. At the end of the fourth week he seemed to be doing very well, but his temperature, which had been approaching the normal, suddenly rose to 104°, accompanying a chill; his belly became enormously distended, his breathing became more and more costal, and he died at the end of the third day from exhaustion and asthenia, with all the physical signs of perforation.

In this connection it is interesting to note that a sudden fall in temperature is not a symptom necessary to the diagnosis of intestinal perforation. On the contrary, there are many cases on record in which a rise of temperature follows this accident. Thus Lereboullet[1] states that in all the cases of perforation he has met with there has been a rise not a fall, and he quotes Lorain, Brouardel, and Thoinot, Griesinger, Amould, Lemoine and Homolle as agreeing with him. Monod[2] also reports such a case.

Dieulafoy[3] goes so far as to assert very positively that peritonitis from perforation very rarely announces itself acutely, with sudden pain and marked constitutional symptoms. On the other hand, its onset is generally insidious. The sensibility of the patient is blunted, the peritoneal infection takes place slowly, and the actual occurrence of perforation may escape unnoticed.

[1] Academie de Médecine de Paris, October 27 and November 3, 1896. Discussion of a paper entitled "De l'Intervention Chirurgicale dans les Peritonites de la Fièvre Typhoide," by Dieulafoy.

[2] Ibid.

[3] Ibid.

While such cases, due to pin-hole perforation, may occur, they cannot be considered common.

Fitz mentions 56 cases in which the onset of symptoms of perforation were severe; 15 in which it was gradual or latent, and 5 in which there was no sign of perforation. Such cases as the last named are recorded by Laboulbéne,[1] who tells us that there was no sign of perforation save a chilliness of the skin and a slight fall of fever. Barth[2] makes a similar report, and Jenner[3] reports a case which left bed on the ninth day and died some hours later of perforation, there being no complaint of pain made.

The diagnosis of perforation is to be reached by the following signs in addition to those just given. Chief and foremost among these is the demonstration of gas in the peritoneal cavity, so that the liver is pushed away from the abdominal wall in such a manner that the ordinary area of liver dulness largely disappears. Percussion of the right hypochondrium is, therefore, an essential procedure in the physical diagnosis of these cases. The only fallacy underlying this test is the possibility of a portion of the colon, when greatly distended with gas, slipping up between the liver and the belly-wall, and thus giving resonance; but this is a rare occurrence. In some cases, however, as already intimated, the symptoms are so insidious, the death so gradual, that a positive diagnosis is not positive, and cases are not rarely seen in which the perforation has not been suspected and is found at the autopsy.

There is one precaution to be taken in cases of suspected perforation which must not be overlooked, namely, that peritonitis may develop from extension of the inflammatory process in the bowel or by reason of the migration of micro-organisms through those parts of the bowel-wall which have been impaired by the ulcerative process. In such cases the pain, swelling, and diaphragmatic paralysis may all be present without being due to perforation, and so closely may the symptoms of perforation be

[1] L'Union Médicale, 1877, xxiii. 389.

[2] Bulletin de la Soc. Anat., 1884, lix. 142.

[3] Medical Times, 1850, xxii. 298.

aped that operation has been performed, with the discovery that no perforation had occurred; thus in a case under the care of Herringham, nothing was found at the section and the patient recovered. Perforation may be simulated by rupture of the peritoneum over a swollen mesenteric gland.

Other causes of peritonitis are necrosis of the mesenteric glands, infarction of the spleen, or the development of abscess in an ovary or Fallopian tube.

Very rarely peritonitis arises from cholangitis, with or without gallstones, and Liebermeister has recorded two cases in which rupture of the gall-bladder with escape of gallstones into the abdominal cavity took place.

What the ordinary percentage of perforation is is in some doubt, but according to Murchison,[1] it is in the neighborhood of 3 per cent. Schulz[2] found it in 1.2 per cent. of 3686 cases of typhoid fever in Hamburg in 1886 and 1887, and Liebermeister[3] in 1.3 per cent. in 2000 cases in Basel in 1865 to 1872. Berg, in 1626 cases, met with it in 2.2 per cent., and this is about the percentage reached by Osler in cases bathed and not bathed.

The most interesting comparative statement as to the frequency of perforations with and without the bath is that made by Mason.

Thus in Boston City Hospital the percentage of perforations in males was 1.4, and in females 1.3, while under the cold bath in Brisbane it was 3.6 per cent. in males, and 1.6 per cent. in females. Liebermeister's statistics, viz., that there were twelve cases of this accident in 973 patients before the bath and fourteen in 1108 after it was introduced, show a very slight difference.

The percentage mortality of this accident is very high. Of 1721 autopsies the percentage was 11.3, according to Murchison. According to Hölscher it was found in 2000 Munich cases 114 times (5.7 per cent.), and in 20 out of 80 of his cases which ended in death. In 4680 cases tabulated by different writers, Fitz found

[1] Continued Fevers of Great Britain.

[2] Centralblatt fur Allegemeine Path. Anat., 1891, vol. ii. p. 289.

[3] Ziemsen's Encyclopœdia, vol. i.

the proportion to be 6.58 per cent., which agrees with Hölscher's statistics.

Hoffmann found that out of 250 deaths in typhoid fever 20 were due to perforation.

Perforation is very much more frequently seen in men than in women. Fitz in 444 cases found 71 per cent. in men and 29 per cent. in women. In 21 cases of perforation in Basel, 15 were men and 6 were women, and Griesinger in 14 cases had 10 men and 4 women. Murchison also found in 24 cases 16 men and 8 women, although the general mortality of the disease among women was slightly higher than among men. So, too, Bristowe, of London, met with this accident in men in 11 cases out of 15, and, again, Nacke[1] collected 106 perforation cases, in which 72 were in men and 34 were in women.

The period of the disease in which perforation most commonly takes place is at the end of the third week or later. Thus in twenty-two cases in which reliable information could be obtained by Liebermeister, perforation took place at the end of the second week twice, during the latter half of the third week, six times, in the fourth week twice, in the fifth week six times, in the sixth and seventh weeks twice each, and later than this twice. Nacke found it 84 times out of 185 cases in the first two weeks, and 99 later, 62 out of 117 cases in the first four weeks, and 55 later.

More accurate statistics are those of Fitz, who in 193 cases obtained facts shown in the following table:

DATE OF OCCURRENCE IN PERFORATION.

	Cases.		Cases.
First week	4	Eighth week	3
Second "	32	Ninth "	2
Third "	48	Tenth "	4
Fourth "	42	Eleventh "	3
Fifth "	27	Twelfth "	1
Sixth "	21	Sixteenth "	1
Seventh "	5		

[1] Ueber die Darmperforation im Typhus Abdominalis. Dissertation, Wurzburg, 1893.

The part of the bowel most frequently perforated in 136 cases was the ileum in 106 cases, the colon in 12 cases, and the vermiform appendix in 15 (Liebermeister). Hoffmann[1] tells us that out of 20 cases the perforation occurred once near the ileo-cæcal valve, four times at four to six inches above, nine times at eight to twenty inches, twice at four and a half to six feet above, once at ten feet above, and in one case there were no less than twenty-five to thirty perforations in the jejunum. In 167 cases collected by Fitz, the perforation occurred in the ileum in 136 instances (81.4 per cent.), in the large intestine in 20 (12.9 per cent.), in the vermiform appendix in 5 cases, in Meckel's diverticulum in 4, and in the jejunum in 2. In 19 cases there were two perforations, in 3 five perforations, and in 4 four. Another case with multiple orifices has been cited.

A very extraordinary case is that reported by Heagler.[2] A woman suffering from ventral hernia was attacked with typhoid fever and perforation of the ileum occurred in the hernial sac. This resulted in sloughing, and a fecal fistula of large size was formed. Great emaciation ensued, but the woman recovered.

An interesting case of typhoid fever with secondary lesions involving the left half of the scrotum has been reported by Spencer.[3] The patient was thought to be suffering from influenza; and had suffered from a hernia in the left inguinal region for nine years. When first seen at the hospital the left half of the scrotum was greatly swollen and distended, the skin being œdematous; the swollen area was tympanitic on percussion, opaque to light, and fluctuated, and at the inguinal region there was a firm mass to which an impulse was transmitted on coughing. An incision was made from which pus, gas, and sloughing omentum came away. The patient died seventeen days later, and the post-mortem revealed the fact that the condition of the scrotum had been due to the perforation of a typhoid ulcer.

[1] Untersuch. und der path. anat. Verand. d. Organe beim Abd. Typhus, 1869.

[2] Correspondenzblatt für Schweizer Aerzte, 1896, No. 17.

[3] London Lancet, April 10, 1897.

In children this accident is very much more rare than it is in adults. J. Lewis Smith states that it is met with only once in 232 cases. Wolberg found no such accident in 277 cases of the disease in children at Warsaw. Fitz gives the following table:

AGE AT WHICH PERFORATION OCCURS.

1	to	10	years	.	.	.	.	.	7	= 3.6	per cent.
10	"	20	"	.	.	.	.	.	46	= 23.8	"
20	"	30	"	.	.	.	.	.	77	= 39.8	"
30	"	40	"	.	.	.	.	.	45	= 23.3	"
40	"	50	"	.	.	.	.	.	14	= 7.2	"
50	"	60	"	.	.	.	.	.	2	= 1.0	"
60	"	70	"	.	.	.	.	.	1	= 0.5	"

In this connection the account given by Taupin[1] of intestinal perforation in children is of great interest. He tells us that he saw two such cases, and that four such were reported in 1834, 1835, and 1838 by Husson and Barrier. Three of these were gravely ill, and when perforation occurred they passed into collapse and died. In the two Taupin saw atrocious pain developed in the right flank and collapse ensued. Death occurred in thirty-six hours, with all the signs of peritonitis.

To one unacquainted with the subject it would seem that there could be no question as to the danger of death from this lesion, yet as short a time ago as 1891 Reeves stated that he had seen five cases presenting all the signs of perforation, and yet the patients recovered. At the same meeting Loomis said he had never seen recovery after the presence of unmistakable signs of perforation. The latter view was that held by most of the earlier writers; but Buhl, in 1857, recorded a case in which death did not succeed perforation for forty-five days, and then as the result of hemorrhage from a mesenteric artery. The autopsy showed that a perforation had occurred, but had been closed. Murchison states that rare cases are met with in which recovery takes place. At the present time it is a well-recognized fact that cases may recover, but that, as Murchison says, they are rare, unless

[1] Journal des Connaissances Med. Chi., 1839.

surgical aid is given the patient very soon after the accident. (See operative interference.)

Perforation does not always produce death, however, because it may not cause anything more than a very localized abscess, owing to a protective peritonitis which walls off the general cavity from infection. Elsner[1] reports such cases, and Pearson[2] records a case in which during relapse an ileocœcal abscess formed, the pus having a fecal odor. In another case[3] a man had a perityphilitis on the twenty-eighth day, and passed two ounces of pus by the rectum on the fiftieth day. Keen records a case in which an abscess formed in the right side, which opened into the ascending colon, and finally a fecal fistula developed. He also records a case sent him by Dr. Schuremen, of Tom's River, N. J., of an abscess which opened near the anus, giving vent to a great deal of pus, in the third week of the disease. Later another opening formed. Major[4] records a case in which collapse occurred on the eighteenth day of the disease, and three weeks later an abscess burst into the rectum, and the patient recovered.

Low's[5] cases had symptoms of perforation in the third week, and peritonitis. Later an abscess burst through the abdominal wall, but the patient recovered. Again, in Lehman's case perforation occurred at the end of the third week, and death occurred a month later. In the abdominal pus the bacillus of Eberth was found. Schmidt[6] has recorded a case of pyopneumothorax subphrenicus, from which three quarts of pus containing a pure culture of the bacillus of Eberth was obtained.

That death does not always follow rapidly after perforation of the bowel in typhoid fever is also proved by the case reported by O'Carroll,[7] in which perforation of the intestine occurred on the thirty-sixth day, and the patient did not die until the fifty-ninth

[1] Transactions of the Medical Society of the State of New York, 1892, 314.
[2] British Medical Journal, 1891, i. 861.
[3] Adam. Australian Medical Journal, 1887, ix. 182.
[4] British Medical Journal, 1891, i. 18.
[5] Ibid., 1881, ii. 122.
[6] Deutsche medicinische Wochenschrift, 1896, No. 32.
[7] British Medical Journal, February 13, 1893.

day, when an adhesive peritonitis was found, and an abscess which had been walled off from the rest of the peritoneum. All of the intestinal ulcers except the one which had perforated had healed. (See also operative interference.)

Without doubt many of the cases of so-called perforation which have been reported as ending favorably have been cases in which there was no perforation, and only a more or less severe localized peritonitis. The symptoms of this condition may be so precisely those of perforation, that an autopsy or exploratory incision may be needed to differentiate them, and peritonitis may arise from so many intra-abdominal lesions that its presence from these causes must always be suspected.

At the present time the prognostic and therapeutic view of cases of perforation are well expressed by the following quotations from Gairdner, Fitz, Keen, and others :

Gairdner[1] says : " What, then, is the proportion of cases which recover without surgical interference when symptoms of general peritonitis have set in ?

" It is difficult to estimate the proportion numerically, but such recoveries are certainly exceedingly rare. Thus, Todd and Jenner,[2] in a long life of large experience, saw one case each ; Tweedie, 2 ; Murchison carefully collected six cases, but only two were his own.

"A fair number of cases may be found in medical literature, reported with more or less accuracy, but it is seldom that an individual experience includes more than one case, while many of large experience have seen no such cases, and even doubt the possibility of recovery after perforation of the intestine freely into the peritoneal cavity. Now, Murchison, at p. 524 of the second edition of his work on continued fevers, states that in ten years, between the publication of the first and second edition of that work, he had attended ' more than two thousand cases ' of enteric fever ; certainly he must have attended even more before the pub-

[1] Glasgow Medical Journal, vol. xlvii. p. 100.

[2] Collected Essays and Lectures on Fevers, pp. 311 and 484, London, Rivington, Percival & Co., 1893.

lication of the first edition; so that his personal experience up to that time may fairly be put down as at least five thousand. In another place he estimates the occurrence of perforation of the intestine in his cases at a fraction over 3 per cent., so that in about 150 of these cases that accident must have occurred. Two only, as we have seen, recovered.

"If, then, the number of unsuccessful laparotomies published be trebled, so as to make sure of including those unpublished, roughly this gives fifty-four unsuccessful cases and five successful cases.

"When it is remembered that little selection has been made in the cases operated on (Van Hook's dictum is, 'the only contraindication is a moribund condition of the patient'), it may be claimed that the 'prentice hand' of surgery has considerably improved on the very best treatment by other means."

Fitz says: "It appears from this statement that of twenty-seven cases of peritonitis in typhoid fever, whatever may have been the cause of the former, though often attributed to intestinal perforation, three recovered after operation, seventeen after resolution, and nine after the spontaneous discharge of the pus. The comparison of this series of cases with those showing the results of early laparotomy for symptoms suggesting typhoid perforation, indicates that the appropriate treatment for this complication would be delay until a probable encapsulated exudation proved unduly slow in absorption. An immediate or early laparotomy for the relief of the peritonitis seems advisable only when the patient's condition is exceptionally good. Should the signs of the exudation persist for a week or more, and the general condition of the patient permit an incision, surgical treatment would then be strongly advisable. That the patient may live for weeks after perforation has taken place is illustrated by the cases of Buhl and Hoffmann already mentioned.

"In brief, immediate laparotomy for the relief of suspected intestinal perforation in typhoid fever, is only advised in the milder cases of this disease. In all others, evidence of a circumscribed peritonitis is to be awaited, and may be expected in the

course of a few days. Surgical relief to this condition should then be urged as soon as the strength of the patient will warrant."

Keen says: "When once physicians are not only on the alert to observe the symptoms of perforation, but when the knowledge that perforation of the bowel can be remedied by surgical means, has permeated the profession, so that the instant that perforation takes place the surgeon will be called upon, and, if the case be suitable, will operate, we shall find unquestionably a much larger percentage of cures than have thus far been reported. But even at present we have a reasonably large number from which to draw conclusions. In the table appended to this chapter Dr. Westcott has collected eighty-three well-authenticated cases. This gives, as a general result, sixteen recoveries, or 19.36 per cent. of cures and 80.64 per cent. of deaths. When this is contrasted with Murchison's unchallenged figures of 90 to 95 per cent. of deaths after perforation without operation, we may well take courage for the future."

Since Keen's essay was published additional cases have been collected by Platt,[1] who says that to Keen's list he is able to add three fatal cases published before 1898, but of which he knows nothing more than the result, fourteen cases which have been recorded subsequently, and his own three cases. The additional cases are as follows: J. H. Nicholas,[2] two cases reported to the Royal Academy of Medicine in Ireland, 1889, both fatal. Podres and Obalinski, cited by Gasselewitsch and Wanach,[3] one fatal case. Gasselewitsch,[4] one case; operation immediately after the onset of symptoms of general peritonitis; perforation sutured; death after forty-three hours. J. B. Deaver,[5] a male, aged twenty-seven years; perforation during the second week; operation within twenty-four hours; free gas in the abdomen; no attempt made to localize the perforation owing to great distention

[1] London Lancet, February 25, 1899.

[2] The Lancet, August 3, 1889, p. 219. [3] Loc. cit.

[4] St. Petersburger Medicinische Wochenschrift, 1898, No. 2 (Centralblatt f. Chirurgie, 1898, No. 19).

[5] American Journal of the Medical Sciences, 1898, vol. cxv. pp. 189–192.

of intestines; irrigation and drainage; recovery; Widal's reaction obtained. H. C. Deaver (reported by J. B. Deaver),[1] a male, aged thirty-six years; perforation in the third week; operation after thirty-six hours; death. Burrell and Bottomley,[2] two cases, one recovery and one death. Handford and Anderson (Nottingham),[3] a male, aged twenty-seven years; perforation on the thirty-third day of a mild attack; operation after twenty-two and a half hours; perforation sutured; peritoneum cleaned by sponges; wound closed without drainage; recovery. Pickering Pick (reported by Rolleston),[4] a male, aged twenty-one years; typhoid perforation of the appendix; the appendix removed; death on the second day. A case mentioned by Dr. Goodall in the discussion upon Mr. Pick's case at the Clinical Society of London: A girl, aged eight years; perforation during relapse; sutured; death after four days. J. B. Deaver,[5] a female, aged twenty-three years; perforation on the twenty-first day; operation fifteen or sixteen hours afterward; perforation sutured; death after two and a half days. Woodward,[6] a male, aged eighteen years; perforation at the end of the second week; operation after nine and a half hours; perforation sutured; the patient recovered from the operation, but died nine days later from typhoid fever. At the necropsy the peritoneum was found uninflamed, with the exception of a local dry peritonitis around the seat of perforation. Cushing,[7] three cases: (1) A male, aged nine years; perforation at the end of the second week; sutured; abdomen opened twice subsequently, once for a supposed second perforation and once for kinking of the intestine from adhesions; recovery. (2) A male, aged eighteen years, perforation in the fifth week; sutured; death in four hours. (3) A male, aged thirty-one years; perforation at the end of the

[1] Loc. cit.

[2] Medical and Surgical Reports, Boston City Hospital, 1898, p. 126.

[3] British Medical Journal, 1898, vol. ii. p. 220.

[4] Transactions of the Clinical Society of London, 1898, vol. xxi. p. 234.

[5] Annals of Surgery, 1898, vol. xxviii. p. 144.

[6] Boston Medical and Surgical Journal, 1896, vol. cxxxix. p. 317.

[7] Johns Hopkins Bulletin, Nov., 1898 (British Medical Journal, Epit., Feb. 4, 1899.)

fourth week; sutured; death after eight hours. In a fourth case no perforation was discovered at the time of operation; the patient recovered. Bigger and Campbell[1] (a case reported to the Ulster Medical Society): A male, aged thirty-six years; perforation during the third week; operation after ten and one-half hours; perforation closed by Lembert's sutures; death on the fourth day. Altogether we have 103 cases, with 21 recoveries. The successful cases on record are as follows: Mikulicz (1884), Wagner (1891), Van Hook (1891), Abbe, Netschajew and Trojanow, Dandridge, Ferraresi (1894), Hill, Murphy, Price (two cases), Watson, Sifton (1895), Finney, Brunton and Bowlby (1896), Panton (1897), Deaver, Burrell and Bottomley, Handford and Anderson, and Cushing (1898). In England, including Platt's own cases, but excluding the doubtful case mentioned by Greig Smith,[2] thirteen operations are now on record. Dr. Platt's own case is the third reported in that country in which the result has been successful.

As this book goes through the printers' hands I find the following case reported by Dr. Hugh M. Taylor,[3] of Richmond, Virginia.

The patient was a "little boy," age not given, who suffered from a typhoid perforation. Operation was performed fifteen hours after the first onset of symptoms. A quantity of seropurulent fluid escaped from the peritoneum but no gas. The perforation was found in the lower portion of the ileum which was closed. The patient recovered, the final report being made four weeks after the operation was performed.

The Russian cases referred to above are, in brief detail, as follows: In 1891 Netschajew and Trojanow operated upon a man aged thirty-one years, who presented signs of perforation. The operation was practised six hours after the entrance of the patient into the hospital. Marked evidences of serous peritonitis were found, and fecal matter was in the peritoneal cavity. Resection of the perforated portion was performed. Death followed, and

[1] British Medical Journal, 1899, vol. i. p. 89.

[2] Abdominal Surgery, fourth edition, p. 776.

[3] Virginia Medical Semi-monthly, March 24, 1899.

autopsy revealed typhoid ulcers in the ileum. In 1893 the same authors operated upon a man of twenty-eight years, who presented similar symptoms. On entering the hospital a diagnosis of the ambulatory form of typhoid fever was made, with intestinal perforation. The operation took place seventeen hours after entrance. A general sero-purulent peritonitis was found, the fluid being filled with fibrinous flakes. Quite a large perforation was present; 4 cm. of the intestine was excised. The abdominal wall was not immediately sutured, but the opening in it was packed with sterile gauze, and complete recovery followed.

A third case was that of Kohltzoff, and occurred in a man of twenty-four years, who had typhoid fever and congestion of the lungs. He had symptoms of perforation, with excessive fever. The operation was performed four hours after the perforation. Sero-sanguinolent fluid was found in the peritoneal cavity, and 20 cm. from the ileo-cæcal valve there was a perforation. A resection of the perforated portion of the intestine was performed. Death occurred in two days. At the autopsy numerous typhoid ulcers were found near the point of ulceration.

In the fourth case, belonging to Trojanow, a patient twenty-nine years of age had been sick for fifteen days with fever. He was seized with violent pain in the belly, followed by intense chills, vomiting, and hiccough. There was abdominal swelling and general pain. The symptoms were those of perforative peritonitis. The operation began sixteen hours after the accident. Abundant sero-purulent fluid was found in the abdominal cavity, and perforation of the intestine had occurred 10 cm. from the ileo-cæcal valve. Resection of the perforated segment was performed, and death occurred fourteen hours afterward. The autopsy revealed typhoid ulcers in the ileum.

In addition to these cases Gasselewitsch and Wanach report five more. The first of these was a man, thirty-six years of age, who had had typhoid fever fifteen days. After eight days he had had bloody stools, violent pain in the belly, followed by intense chills, vomiting, and hiccough. The belly was swollen and the pain was general. The pulse was 120, the temperature febrile. An operation

was performed two hours after the perforation. On exploring the intestines two perforations were found, one 2 cm. in diameter, the other much smaller. Twenty centimetres of the intestine was resected, and death followed in about two hours. At the autopsy ten ounces of fetid pus was found in the belly. The parietal and visceral peritoneum were covered with punctiform hemorrhages. The part of the intestine resected was 37 cm. from the cæcum. Their second case was a man of twenty-four years, who had been ill some time with typical typhoid fever. Seven days after entrance into the hospital he was seized with violent chills and fever and all the symptoms of perforative peritonitis. The operation was done seventeen hours after the accident, ether being given after a preliminary injection of cocaine. Perforation of the intestine was found. The mesenteric glands were enlarged and were adherent to the intestine in places. Thirty centimetres of the intestine was resected. Death occurred in six hours after the operation. The autopsy revealed profound typhoid ulceration at the lower extremity of the ileum. There were also signs of catarrhal pneumonia. The third case was in a young man of nineteen years, who had been sick five days. His fever was high, and he had bloody stools. Four weeks after his entrance into the hospital he had perforation of the intestine. His condition remained grave, and on opening the peritoneal cavity it was found to be filled with bloody fluid, and there were intestinal adhesions. Death occurred in three days. Again, the autopsy revealed perforation and ulceration. A man of twenty-seven years presented mild symptoms of typhoid fever. Six days after his entrance he was seized with violent pain in the belly and with chills and sweating. There was also meteorism. Twenty-four hours after these symptoms the operation was performed. Again the belly was found filled with sero-purulent fluid. Thirty centimetres of the intestine was removed and contained four ulcers. Notwithstanding injections of saline solution the patient died eight hours after operation. Again the autopsy confirmed the diagnosis. In the fifth case a man of twenty-nine years entered on the seventh day of typhoid fever; six days later violent pain in the cæcal

region came on, with moderate fever. Surgical intervention took place thirteen hours after the accident. The abdominal cavity was filled with serous fluid. The walls of the intestines were œdematous. Resection was performed. Death occurred in three days. The autopsy revealed the characteristic lesions and pneumonia of both bases of the lungs. Altogether, these authors quote seventy-one instances of perforation in the course of typhoid fever, with seventeen recoveries.

The number of deaths in operation for perforative peritonitis in typhoid fever is necessarily high.

In summing up his views on the question of operative interference, Keen says: "Mr. Gairdner, Assistant Physician of the Belvidere Fever Hospital, in analyzing forty-seven cases of peritonitis in typhoid fever with reference to surgical interference, in a very careful and judicious paper in the *Glasgow Medical Journal*, February, 1897, page 67, reaches the following conclusions, which well express my own feelings, and it is all the more worthy of consideration as the opinion of a thoughtful physician rather than that of an over-sanguine surgeon: 'The treatment of peritonitis in the course of enteric fever by laparotomy has hitherto had a moderate success. There is every reason to believe that greater success is possible, and in any case the results are better than those of any other treatment. Laparotomy offers a fair chance to about 49 per cent. of cases, while 19 per cent. of the whole would certainly have a good chance. Nothing but experience can determine what the results will be –better or worse than might be expected *prima facie*. If there is a good cause at least for attempting surgical interference, it becomes incumbent on the profession to afford every facility for making the attempt. This, of course, applies particularly to authorities responsible for hospitals in which enteric fever is treated.'"

My own feeling in this matter is well summed up in the words of Mikulicz,[1] who said at Magdeburg in 1884: "If suspicious of a perforation one should not wait for an exact diagnosis

[1] Quoted by Thayer in Progressive Medicine, 1899, vol. i.

and for peritonitis to develop to reach a pronounced degree, but, on the contrary, one should immediately proceed to an exploratory operation, which in any case is free from danger." Again, Cushing[1] says: "When the diagnosis is made, operation is indicated whatever the condition of the patient. As Abbe's case exemplifies, no case may be too grave. A precocious exploration from an error in diagnosis is not followed by untoward consequences, such as must invariably be expected after a neglected and tardy one." The only modification I would make of these two opinions is that in sudden acute cases followed by speedy collapse, the patient should be given sufficient time to rally before the operation is performed.

The relation of typhoid fever to *appendicitis* is one of great interest. It has been thought by some that appendicitis arising in typhoid fever was a mere coincidence; by others that its origin depended upon a general infectious process, and, again, by others, that it was due to the direct infection with the bacillus of Eberth. Probably all these views hold true in individual cases. The richness of the appendix in lymphoid tissue, and the fact that typhoid fever is particularly prone to attack such tissues, renders this organ peculiarly susceptible on theoretical grounds. That this view is correct is proved by the research of Hopfenhausen,[2] who preserved the appendices obtained from thirty cases of typhoid fever and studied them under Stilling in the University of Lausanne. She concludes that moderate changes in the appendix may be found in nearly all cases of this character, and that it is most marked in the earlier stage of the malady, and consists chiefly in cellular infiltration, specific lesions being rare and not being sufficient to produce the more severe forms of appendicular disease.

True appendicitis complicating typhoid, in the sense of inflammation of this part severe enough to produce abscess, is undoubtedly a very rare affection. One such case is reported further on,

[1] Johns Hopkins Hospital Bulletin, 1898, vol. ix. p. 257.

[2] Revue Méd. de la Suisse Romande, February 20, 1899.

as occurring in my own practice. Here a large abscess containing over a pint of pus, having the odor of a typhoid-fever stool, was allowed to escape by an incision. Recovery occurred. In more frequent instances the appendix is the seat of typhoid ulcers, or an ulcer, although the recorded cases in which this lesion has been found are surprisingly few. This scantiness of reports is probably due in large part to the fact that the appendix is not carefully examined for lesions in making autopsies, for in the cases with which I am acquainted in which the appendix has been carefully examined, appendicular lesions have been surprisingly frequent. At a recent meeting of the Pathological Society of Philadelphia Stengel made a verbal report of several instances in which typhoid ulcer had been found in the appendix, as did also Sailer, and in a paper on typhoid ulcer of the œsophagus, Riesman incidentally mentioned appendicular typhoid ulcer as being also present in his case.

Keen has well said, therefore, in his essay, that in all cases of operation for intestinal perforation in typhoid fever the surgeon should examine the appendix to discover if it is diseased. In Keen's table of operations done for intestinal perforation, cases of associated appendicular lesions are recorded by Bontecou,[1] Kimura,[2] and Alexandroff[3] (there were three large perforations of the appendix in this case).

Additional cases have been chiefly collected by Kelynack,[4] who points out that Murchison[5] saw two cases of appendicular ulceration, one in a girl of thirteen years, four ulcers being present. Two small perforations were found in it. Norman Moore[6] records four cases. Death was due in two of them to perforation of the appendix; another had an ulcer at the tip of the organ. Fitz found in 257 cases of appendicular perforation only three due to

[1] Journal of American Medical Association, January 28, 1888, p. 106.

[2] Sei-i-kwai Medical Journal, 1890, ix. 55.

[3] Report of Hospital St. Olga, in Moscow, 1890, 198.

[4] Pathology of the Vermiform Appendix, London, 1892.

[5] The Continued Fevers, 2d ed., 1873, p. 623, and Trans. Pathological Society, London, 1866, xvii. p. 127.

[6] Trans. Pathological Society, London, 1883, xxxiv. 113.

typhoid fever, and in a later paper,[1] in 167 cases five instances with this lesion. All these quotations throw light on this matter, but the reports of Morin[2] and Heschl[3] give a much higher percentage. Thus Morin, in 67 collected cases, finds 12 examples of appendicular perforation, or 18.75 per cent., and Heschl, in 56 cases, found this lesion in 8, or 14.3 per cent. McArdle[4] has also reported a case.

Contrary to the view held by some, that perforation of the appendix often occurs in typhoid fever, it is to be recalled that Fitz in one of his early investigations was only able to find three cases in which this accident occurred as the result of typhoid fever. More recently Fitz has collected five cases in 167 cases of perforation due to typhoid fever. On the other hand, perforation in this part is more apt to be followed by recovery than elsewhere, and this may explain why it is that the best post-mortem records are so scant in this respect. Fitz asserts that the more closely the symptoms of perforation resemble those of appendicitis the more favorable is the prognosis.

Rolleston[5] states that in 14 out of 60 cases of enteric fever seen at St. George's Hospital, London, changes were found in the appendix. In 5 there was tumefaction, in 7 ulceration, and in 2 perforation. Perforation of the bowel occurred in 18 of these 60 cases—a very high percentage.

In the very interesting paper by Hopfenhausen[6] on this topic, already quoted, she tells us that she collected statistics concerning the appendix in 808 cases which came to autopsy in St. Petersburg,[7] and found perforation of the appendix in eight cases. In one of these the perforation had caused perityphlitis, found post-mortem; in two others the diagnosis was made in life. In

[1] Trans. of Association of American Physicians, 1891.

[2] Thèse de Paris, 1869. [3] Schmidt's Jahrbucher, 1853, lxxx. p. 42.

[4] Trans. Royal Academy Medicine, Ireland, 1888, vi. 392.

[5] Lancet, 1898, vol. i. p. 1401.

[6] Revue Méd. de la Suisse Romande, February 20, 1899. Étude sur l'état et l'appendice vermiforme dans le cours de la fièvre typhoide.

[7] Protocoles des instituts pathologique de l'Hôpital Municipal d'Obouchow, et de l'Hôpital Municipal de Ste Marie-Madeleine, 1889–1897.

117 cases general peritonitis was found, and in 109 this was attributed to intestinal perforation.

In all probability typhoid fever predisposes a patient to appendicitis. Keen has hinted at this without adducing any statistics to prove it, and cases can be found in literature which point to it. In the cases collected by Hopfenhausen,[1] we find this subject also discussed. She found the following statistics:

	No. of cases proceeding from typhoid fever.	No. of cases observed.
Hôpital cantonal de Lausanne	9	200
Sonnenburg	6	130
Pozzi	1	1
Bull	3	12
Hecker	1	35
Bossard	2	26
Douneff	4	52
Le Guern	1	110
Jacobson	2	6
Schnellen	1	32
Langheld	4	112
Hohn	1	2
Jacob	2	25
Total	37	743

The interval between the two diseases in these cases was generally so long that the figures disprove the relationship rather than prove it. Thus, in 5 cases it followed in from twenty-five to forty years; in 24 from ten to twenty years; in 2 cases in three years; in 1 in two years; in 1 in one year; in 3 from three to six months; in 1 during typhoid fever.

In only one instance was the appendicitis near enough to the attack of typhoid fever to bear the true relationship of cause and effect, namely, that of Bossard,[2] in which perityphlitis followed in the same month.

[1] Revue Médicale de la Suisse Romande, February 20, 1899.

[2] Uber die Verchwärung und Durchborung des Wurmfortsatzes. Thesis, Zurich, 1869.

The history of my own case, to which reference has already been made, was as follows : On March 23, 1898, J. R., aged forty-three years, called on me with the statement that he was suffering with general malaise and aching all over the body, and thought that he must have caught a severe cold. Two days later, March 25th, I was sent for to go to his house, and found him with a temperature of 102°. He also complained of a little more soreness upon the right side of his body than upon the left, but this was not particularly localized. As he had a history of an obscure attack of appendicitis eighteen months before, an attack in which he asserted that there was swelling but no pain in the right iliac fossa, I made a careful examination of the region of the appendix, but was unable to discover any induration and but slight tenderness, with a good deal of gurgling. His temperature from this time continued to rise, and the pain in the neighborhood of the appendix increased, but at no time was it very severe.

On the 26th I asked Dr. W. W. Keen to see him with me in consultation, but neither of us could determine that there was any inflammation of the appendix. The fever continued high, his condition became worse, and on Monday, April 4th, I asked Dr. Musser to see him with me in consultation, as there had developed in the right iliac region an increased tenderness, some pain on extension of the leg, and the patient was unable to lie upon that side. Nothing connected with the appendix could, however, be discovered, but as there was considerable bulging in a line drawn between the axilla and the anterior superior spine of the ilium, and as this swelling evidently contained pus, it was decided that an operation was needed, and I asked Dr. Keen to see the case. Dr. Keen agreed in the diagnosis, and on Saturday, April 8th, he made an incision from which escaped about a pint and a half of exceedingly offensive pus, with a distinctly typhoidal odor. The stools prior to the operation had been somewhat typhoidal in character, the odor of his body was that of typhoid fever; he had developed a number of rose spots on his abdomen and back, and the appearance of his tongue was characteristic. An examination of his blood before operation revealed a considerable

number of leucocytes, probably due to the abscess formation, and gave an imperfect Widal reaction. In the abscess cavity there was found a considerable mass of tissue about the size of my thumb which was at first thought to be a sloughed off appendix, but which on careful examination by Dr. Keen apparently consisted of a piece of omentum which had been cut off from the general peritoneal cavity.

After the operation the patient's temperature rapidly fell so that it reached the normal point in a few days, and while he was exceedingly ill for some days prior to and after the operation, he ultimately made a perfect recovery.

An inoculation was made from the appendix on April 16th. The tube bore the date of April 9th, and contained a growth at the time it was received. Stains of the growth and of subsequent cultures showed a short thick bacillus with rounded ends, usually single, some holding together in pairs or short threads of three to six bacilli. Also a few bacilli about the same length as the above, but much thinner and with a tendency to form longer threads. They stained readily with the ordinary aniline dyes. If stained for only two minutes little light granules on the side and near the end were observed, but if stained longer they also reacted to the stain. These spots failed to react to stains for spores, and were probably granular areas. By their growth and reaction to stain they correspond to the bacillus coli communis and proteus vulgaris.

A second case without abscess was that of a boy of nine years, who because of ill health had been taken to the seashore, with the hope that it would benefit him. During the first week at Atlantic City he suffered from continued fever, ranging from 102° to 103°, for which no adequate cause could be discovered. His fever then disappeared suddenly, and was absent for a week, during which time he ate heartily and seemed to improve greatly in health. During his third week at Atlantic City, however, the fever returned in an irregular form, and he complained at times of violent pain in his abdomen. Two days after his return to Philadelphia I saw him. At this time there was marked tenderness

in the right iliac fossa, particularly in the neighborhood of McBurney's point, and also posteriorly, back of the appendix. There was also some rigidity of the muscles on the right side over the appendix. His temperature varied from 103° to 104°, but he was not particularly restless. His tongue was fairly clean, but there was a complete loss of appetite. At this time, the appendicular trouble did not seem sufficient to account for his high temperature, but a careful examination of every organ of his body and of the blood failed to reveal any cause for the pyrexia. At the end of the first week in bed his tongue became foul, his lips covered with sordes, the temperature on one or two occasions rose nearly to 105°, and he developed the typical rose spots of typhoid fever, the appendicular irritation and inflammation having been treated during the preceding week by the application of ice-bags. One week after the symptoms of typhoid fever became well marked, distinct appendicular tenderness partly disappeared, and at the end of the third week had entirely disappeared.

These two cases are of interest because they illustrate the fact that it is sometimes necessary to make a differential diagnosis between typhoid fever and appendicitis, and also because they illustrate the fact that typhoid fever and appendicitis may exist side by side. Possibly in one or both of these cases the appendicular trouble arose from the typhoidal affection.

Nervous System in the Developed Stage of the Disease. Delirium. The nervous disturbances of this period vary very greatly. In the average case there is in the early part of the onset no mental change, save that of unfitness for mental occupation, with dreamful sleep which is apt to be restless. Later the patient continually doses off, yet awakens easily, and for a moment may be a little confused between the mental impressions left on his brain by the dream and the conditions he finds about him on returning to consciousness. Still later, if the attack is marked, he becomes more apathetic when awake, less easily aroused when asleep, and often delirious in his sleep, his dreams being evidently vivid, so that he keeps muttering the conversation he thinks he is actually having, or calls out loudly, as his dream seems to lead him to a

point where an imperative call or sudden action is needed. Sometimes the delusions in the delirium amount to imperative conceptions, and the patient believes that he is away from home and must return there at once, or that he is being restrained by force, or, again, that some member of his family is in distress and needs his aid or is calling for him. Often in parents and in young persons this form of mental disturbance is painful to witness, difficult to overcome, and harassing to the patient. In these cases the hands may be moved continually in active motions, as if to illustrate the views of the patient. Such cases are apt to be grave if for no other reason than that they exhaust themselves if relief is not given. The more encouraging type of delirium is of the quiet, muttering form, as if the patient was gently "speaking in his sleep" as in health, and this may be taken as the natural form of delirium in the disease. Later the stupid condition becomes more and more marked in some cases, and absolute mental stillness is reached, in which only hard shaking or loud calling will arouse the patient.

On the other hand, even in severe cases the mental state often remains but little disturbed throughout the entire illness, and in the majority the beginning mental apathy is largely put aside by the proper use of cold sponging or plunging.

Aside from the mental hebetude of most cases of typhoid fever which may be considered to represent the ordinary mental signs of this disease, we may have remarkable clearness of intellect, so that at no time, even when waking from a heavy sleep, is the patient's mind clouded. On the other hand, it is a curious fact that some of these patients who seem to be mentally clear all through an attack, state after it is over that they have a very indistinct recollection of the occurrences that took place.

There can be no doubt that, as a rule, the mental state is a fair index to the severity of the malady, and, therefore, the greater the perversion of the mental process the more grave the prognosis. So far as delirium itself is concerned, Liebermeister found that in 983 cases without noteworthy brain symptoms only about 3.5 per cent. died; that in 191 cases with mild delirium at times, 19.8

per cent. died, and in 43 cases in which stupor or coma was present, 70 per cent. died. Delirium is a grave symptom in typhoid fever in direct proportion to its severity. Zenner[1] asserts that in cases of severe delirium the mortality reaches 50 per cent., and when the delirium is complicated with stupor, almost 70 per cent.; that the mortality of initial delirium approximates 30 per cent., while that occurring during the first week of the fever is over 40 per cent. It seems to the writer that these statistics give a false impression as to the danger of these symptoms of the disease. These figures, however, express the gravity of marked mental symptoms, and also throw light on the relative frequency of the mild and severe affections of the brain.

Delirium is largely dependent upon the susceptibility of the individual to the infection and to the febrile movement. Many persons are readily made "flighty," to use the popular term, by fever of less than 103°, while others withstand greater fever than this with impunity. A delirium in a child, of the active talkative or complaining type, does not possess grave significance if the fever be high enough to be its cause, since the mental disturbance is probably due to the temperature, or if this symptom occurs in a nervous woman or man it is not of great importance unless it be so persistent and long continued that the loss of sleep and rest exhausts the patient.

A form of delirium, usually seen in hysterical women and children, which resembles the condition of the patient suffering from belladonna poisoning, sometimes occurs, in which there is much restlessness and tossing of the body, with great volubility and incoherent screaming, which may seem most alarming, but which is not as dangerous an omen as its severity would indicate. As it is usually seen in the early stages it in no wise is indicative of profound nervous exhaustion, but rather of an ill-balanced nervous system upset by the nervous disturbance of the infection.

In severe cases that condition of ceaseless mental activity in a

[1] American Lancet, January, 1889.

semi-stuporous mind, called "coma vigil," is often present. It is an indication of grave infection, as a rule.

Strümpel asserts that "actual insanity is not infrequent during the course of typhoid fever," and that it generally takes the form of a melancholia. Taty[1] records a case of what he calls the melancholic form of typhoid fever, the diagnosis being confirmed by the Widal action and other characteristic symptoms. The patient was restless, had loss of appetite, was delirious upon anarchistic questions, and had great mental depression. There was absolute mutism when she was examined, and she refused both food and drink, but sleep was relatively good. In another case there were visual hallucinations and delirium, with melancholic conceptions, and vague ideas of persecution. Strümpel also records a case of hysterical insanity in a young girl, which broke out during the course of the fever. (For post-typhoid insanity, see last chapter, by Dr. Dercum.)

Hysterical convulsions have been recorded as complicating the developed stage of typhoid fever; thus Rémond and Coumenges[2] record two cases of this character. In one, a young woman of distinctly neurotic character, who had never suffered from convulsions however, developed on the fifteenth day of the disease unconsciousness, a thready pulse, embarrassed respiration, and severe hiccough, so that the physician thought the patient was about to die, when the scene suddenly changed, the body was stiffened, and a violent hysterical convulsion came on. Repeated attacks occurred on subsequent days until death occurred from exhaustion.

The *headache*, usually frontal and severe, in the early days of onset, may continue as an annoying symptom all through the attack, but rarely possesses its severe characteristics after the first week. Under certain circumstances, however, it becomes severe, and is worthy of relief and study, since it may be due to periostitis of the skull, to abscess of the middle ear or brain, or to uræmia. A combination of more or less active delirium with rest-

[1] Lyon Médicale, 1897, p. 291. [2] Medical Bulletin, June, 1895.

lessness and disturbed sleep and severe pain in the head should make a careful search for a local cause necessary. In other cases the pain extends from the head down the spine, even to the sacrum, and from there down the legs, particularly along the posterior parts and in the bones. This pain is chiefly seen in onset and in the early stages, and is generally absent by the third week.

Hysterical symptoms may be present in children. Thus De Witt[1] reports the case of a boy of twelve years, who suffered on the twenty-third day from marked hysterical symptoms, supra-orbital neuralgia, and pain and stiffness in the back, the symptoms coming on simultaneously with high temperature.

Meningitis. Rarely in the course of typhoid fever of the uncomplicated form symptoms of irritation or inflammation of the meninges of the brain develop, and it is important to remember that these symptoms may arise from several causes. The most common of these is congestion and engorgement of the meningeal vessels without any true inflammatory process, the next most common form is that due to the extension of an infection from abscess in the middle ear; the third form is that in which there is infection with the streptococcus or pneumococcus, and very rarely do we find a meningitis due to the bacillus of Eberth. Osler records three cases in which he made autopsies in suspected typhoid meningitis and found no true inflammation, and as long ago as 1839 Taupin called attention to the difference at autopsy between the appearance of the meninges of the brain in death with meningeal symptoms due to typhoid fever and those due to true meningitis. In typhoid fever in children he states that the condition is one of effusion without hyperæmia.

Keller[2] asserts that true meningitis in a child can be differentiated from typhoid fever with meningeal symptoms by the fact that "Kernig's sign" is present in meningitis and absent in enteric fever. This sign consists in placing the child in the position of dorsal decubitus with the legs in complete extension. After

[1] Bulletin de l'Academie Royal de Médecine de Belgique, November 17, 1889.

[2] Revue des Maladies de l'Enfance, September, 1898, p. 450.

this is done the patient is raised to the sitting posture, when if meningitis be present the knees become flexed and cannot be straightened until the child is once more flat upon its back.

Meningitis in children complicating typhoid fever was written upon as long ago as 1825 by Senn,[1] of Geneva. Three of his cases are evidently cases of typhoid fever, while in others there is doubt as to their authenticity, and there is still less evidence that real meningitis was actually present, even though the symptoms were those of meningeal irritation.

These meningeal symptoms vary greatly in their severity according to the meningeal lesions which may be present. In the majority of instances the chief signs are headache, delirium, some muscular rigidity, particularly in the neck, and, it may be, "lead-pipe" rigidity in the arms and legs. In other instances the patient is too deeply stupefied by the poison of the disease to complain of headache, but may show its presence by rubbing his hands over his head and groaning, after which he may pass into coma, which deepens until death occurs. Very rarely does the pure symptom-complex of true acute meningitis develop, and until the characteristic squint, retraction of the head and pupillary signs are present, the physician must not hasten to a diagnosis of meningitis.

On the other hand, the symptoms already named may be so typical that if the patient is brought to a hospital late in his illness without a history, he may present so little of the typhoid appearance and so much that of meningitis that a mistake in diagnosis is readily made. To quote Hirt,[2] "Of all diseases typhoid fever is most likely to be taken for meningitis," and, again, he tells us that "we might believe that at least the characteristic temperature-curve, the splenic enlargement, and the rose spots would be sufficient to make a mistake impossible;" but this is by no means always the case; there are instances in which typhoid fever cannot with certainty be excluded, and then the differential diagnosis is simply impossible.

[1] Recherches sur la Meningite Signe des Enfants, 1825.

[2] Nervous Diseases, American edition, p. 18.

So certain, however, is Money[1] of the assertion of Hughlings Jackson, that the knee-jerk is not absent in typhoid fever, that he uses this sign as a point in differential diagnosis. Thus in tubercular meningitis he states that it disappears and then reappears every few days, and that this inconsistency of the reflex favors the diagnosis of tubercular meningitis rather than typhoid fever.

The possibility of confusing meningitis or, rather, meningeal symptoms with those of typhoid fever was, however, discussed by Taupin in 1839, and he points out that in such cases the patient has in meningitis due to typhoid fever no convulsions, no strabismus, and no paralysis, whereas the child with true meningitis has all these signs, and in addition a variable pulse, a scaphoid belly, an absence of pulmonary catarrh, and a face which is alternately red and pale.

As an illustration of the rarity of true typhoid meningitis, however, it is of interest to note that from 1855 to 1887 there are only five cases of this affection referred to in the *Index Catalogue* of the Surgeon-General's office, and as none of these were tested bacteriologically they cannot be considered *bona fide*. That meningitis due to any cause in typhoid fever is rare is shown by the fact that out of 2000 cases in Munich, only eleven are recorded as suffering from meningitis. Still more rarely is the meningitis due to the bacillus of Eberth, for Wolff,[2] in 174 cases of typhoid fever which were subjected to bacteriological examination, only found 2.87 per cent. in which the specific bacillus could be found in the meninges. Within the last two years this subject has been admirably discussed by Ohlmacher,[3] of Ohio, and by Keen,[4] of Philadelphia. Ohlmacher himself records two cases in which during the course of typhoid fever meningeal symptoms developed, and in which careful bacteriological research revealed beyond all doubt the bacillus of Eberth in the meninges. In still another

[1] The Lancet, 1889.

[2] Berliner klinische Wochenschrift, 1897, No. 10.

[3] Journal of the American Medical Association, 1897, p. 419.

[4] Surgical Complications of Typhoid Fever.

case recorded by Ohlmacher there was found a mixed infection by this bacillus and the streptococcus.

Altogether but sixteen cases of true meningeal infection by the bacillus of Eberth of an undoubted character have been recorded, which is a point of great interest.

In all of these the dura mater and pia mater appear to be equally affected, and the effusion was in at least six of the cases purulent.

Illustrative cases of this character are taken as follows from Ohlmacher's paper:

"A case of meningitis occurring in the course of typhoid fever was described by Kamen[1] in 1890, in a soldier who entered the hospital after having been ill for five days. A severe headache set in three days later, followed by delirium and unconsciousness, and death occurred eight days after admission to the hospital. Aside from acute splenic tumor and a single typhoid ulcer near the cæcal junction of the ileum, the post-mortem examination showed an extensive purulent leptomeningitis. The cultures obtained from the spleen, mesenteric glands, and meninges were identical, though only the potato test was mentioned as having been employed for identification. The following year Fernet[2] reported the case of a woman who developed headache, delirium, strabismus, exophthalmus, retention of urine and irregularity of the pupils in the course of typhoid fever. At autopsy the characteristic changes of typhoid fever were found in the abdominal cavity, and a diffuse serous meningitis was also present. It is claimed that typhoid bacilli were isolated from the meningeal fluid, though no mention is made of special tests. Silva[3] likewise observed at autopsy in a female epileptic, ten years of age, a sero-hemorrhagic leptomeningitis with a lobar pneumonia and the ordinary evidences of typhoid fever. Typhoid bacilli were isolated from the meninges and carefully identified. Still another case was reported by Honl,[4]

[1] International Klin. Rundschau, 1890, vol. iv. No. 3, p. 98; No. 4, p. 156.

[2] Le Bulletin Médical, 1891, p. 653.

[3] Riforma Medica, 1891, vol. iii. No. 210.

[4] Centralblatt für Bacteriologie, 1893, Bd. xiv. p. 767.

who found a diffuse purulent leptomeningitis in a twenty-one-year-old woman, who died in the course of typhoid fever. An exhaustive differential examination showed the only bacterial species obtained from the meningeal exudate to be bacillus typhosus.

"Cases essentially similar to those just noted have been reported since 1892 by Vincent,[1] Hintze,[2] Mensi and Carbone,[3] Stuhlen,[4] Tictine,[5] Kühnau,[6] and a second one by Kamen.[7]

"Tictine reported two cases which came under his observation, and he also produced a purulent meningitis in animals by means of subdural inoculations with typhoid cultures. The second one of his cases differs from all others in that the patient was perfectly conscious during the last week of his life.

"Profound unconsciousness, delirium, coma, and often retention of urine are the symptoms most often described in these cases. Other symptoms which might suggest an actual meningitis are usually insignificant, and can scarcely be looked upon as of diagnostic import. To this rule, however, the case mentioned by Mensi and Carbone is a notable exception. Their patient was a girl six years of age, who had been ill nine days before entering the hospital. The patient ran the course of a moderate attack of typhoid fever, reaching the stage of apyrexia four weeks after coming to the hospital. Four days later a violent chill occurred, with intense headache and a temperature of 39.2° C. Delirium, opisthotonus, contractions, amblyopia, and dilated non-responsive pupils were successively noted, together with a herpes labialis, paresis of right face, and retraction of abdominal wall. Great prostration followed, and death occurred four days after the onset of this relapse. The autopsy showed a fibrino-purulent cerebro-spinal meningitis, with dilatation of the lateral ventricles, and a bronchitis of the medium and smaller bronchioles. Numerous

[1] Schmidt's Jahrbucher, 1893, Bd. ccxxxvii. No. 2.
[2] Centralblatt für Bacteriologie, 1893, Bd. xiv. No. 14.
[3] Riforma Medica, 1893, vol. i. p. 14.
[4] Berliner klin. Wochenschrift, 1894, No. 15.
[5] Archives de Méd. Experiment, 1894, tome vi. p. 1.
[6] Berliner klin. Wochenschrift, 1896, No. 25.
[7] Centralblatt für Bacteriologie, 1st abtheilung, 1897, Bd. xxi. Nos. 11–12.

typical typhoid ulcers in the stage of healing were found in the ileum and colon; the mesenteric glands were swollen and soft, and there was softening of the spleen. A thorough bacteriological examination of the meningeal exudate resulted in finding typhoid bacilli as the sole bacterial inhabitant."

In rare cases where death has occurred from meningitis without enteric fever being suspected, the autopsy has revealed the bacillus of Eberth to be its cause, as has been reported by Curschman. Such instances have been recorded by Ohlmacher and are of interest. He tells us that:

"In the course of a study of meningitis, Neumann and Schaeffer[1] (1887) found an extensive purulent leptomeningitis in a woman brought to the hospital unconscious, and who died in a few hours without furnishing any history. No lesions of typhoid fever were found, but pure cultures of a bacillus were obtained from the meninges, and these, the authors were led to believe, were of bacillus typhosus, from the general character and from the positive results of the potato and fermentation differential tests. A very similar case was reported soon after by Adenot,[2] in which a woman presented profound symptoms of cerebral infection and died in eight days. Absolutely no typhoidal lesions were present in the intestines, spleen, and mesenteric glands, but from the sero-purulent exudate in the soft meninges a bacillus resembling the typhoid organism was obtained. The only differential test here applied was the growth on potato, and we now know that this is not sufficient to identify the bacillus of typhoid fever. The case recorded by Balp[3] also belongs in the same category with those of the authors just noted. He found a diffuse purulent meningitis in a patient dying five days after a fracture of the skull, and in the exudate a bacillus resembling the Eberth organism was found, together with a species of diplococcus. The phenol and indol tests are all that Balp mentions having used for differentiation."

[1] Virchow's Archives, 1887, Band. cix. Heft. 3, p. 477.

[2] Archives de Méd. Experiment et d'Anat. Pathol., 1889, tome i. p. 656.

[3] Rivista Generale Ital. et de Chir. Med., 1890, No. 17, p. 406.

A case of purulent cerebro-spinal meningitis complicating typhoid fever has also been reported by Stuhlen.[1] The patient was a man whose wife and children were also sufferers from typhoid fever. He first suffered from wretchedness, headache, chills, and constipation. When admitted to the hospital, four days later, there was stupor, restlessness, and delirium, and on the fifth day sudden collapse, from which he rallied, but persistent stupor remained. On the seventh day there was rigidity of the neck and slight jaundice. An examination of the cerebro-spinal pus showed the typhoid bacillus.

Very recently Kerr and Moffitt[2] reported to the California Academy of Medicine the case of a man of twenty-eight years, who on admission was found in a stupid mental state when he had been ill for a period of three or four weeks. He had been seized with general weakness, fever, loss of appetite, headache, and pain in the right iliac region, no cough or nose-bleed. The cause of his entrance to the hospital was the pain in the right iliac region, weakness, and headache. He was found to be slightly demented, and answered questions slowly, articulating poorly, but there was no real aphasia. The fever ran an erratic course, resembling tubercular meningitis more closely than typhoid fever. The pulse was fairly slow and dicrotic. There were no spots and no eye-symptoms; there was persistent diarrhœa of the pea-soup variety, and rapid emaciation; the Widal test was obtained, and autopsy showed a few old ulcers in the right ileum which were certainly six or eight weeks old; the brain was covered with a thick purulent exudate, yellow-red in color. Cultures were made which showed mobile bacilli giving the negative glucose test, but clumping with typhoid serum.

Boden[3] reports the case of a fourteen-year-old child who suffered from typhoid fever and was admitted to the Augusta Hospital of Cologne, on October 2d, at about the end of the first week of the disease. There was hyperæsthesia of the entire body, and

[1] Berliner klin. Wochenschrift, April 9, 1894.

[2] Journal of the American Medical Association, March 18, 1899

[3] Münchener Medicinische Wochenschrift for February 28, 1899.

cyanosis. Two days later there was a severe epileptic attack and deep stupor, with left-sided abducens and facial paralysis, with loss of pupillary reflex and the patellar reflex. Death occurred three days later, and the autopsy revealed marked typhoid fever of the first week, and meningitis serosa, a large amount of clear serum being present at the base of the brain. The brain was normal, the ventricles were distended. From the fluid in the ventricles a pure culture of the bacillus of Eberth was obtained; this fluid also gave the Widal test. Boden states that only five cases of this character have been reported, namely, those of Stuhlen, Kugnan, Daddi, Hintz, and Honl.

The frequency of this complication when due to true typhoid infection of the meninges in the different periods of the disease is in direct ratio to the length of the malady, namely, in the third or fourth week, and in the great majority of instances in which the complication has appeared the patient was under thirty years, and usually between twenty and thirty years, the period in which typhoid fever is most commonly seen.

In every case of true typhoid meningitis, so far as recorded, death has occurred, but this is a statement which does not possess as great prognostic value as would appear at first glance, since an absolute diagnosis of true typhoid meningitis cannot be made during life, for the positive test is the bacteriological examination of the skull contents. Nevertheless, the presence of marked meningeal symptoms is of the gravest import in all cases.

Sometimes, because of degenerative changes in the vessels, a hemorrhagic effusion into the meninges of the brain takes place, but this does not commonly produce marked symptoms unless it is profuse.

Cerebral Thrombosis and Embolism. Richardson[1] has recorded a case of a man of forty-three years, who in the third week of the disease suffered from intense headache, chiefly in the left temporal region, accompanied by collapse and a subnormal temperature. He rallied under stimulating treatment, but two

[1] Journal of Nervous and Mental Diseases.

days later there was marked coma, contracted pupils, particularly that on the right side. Convulsive movements were also present on the left side, chiefly in the leg. Later the right side of the body was involved. He died five days after this complication arose, and the autopsy revealed no signs of meningitis, but the veins of the pia mater were distended with five clots, one of which was particularly large and lay along the Rolandic fissure. The sinuses were patulous. In the first left temporal convolution there was a small abscess. No clots were found in the sinuses. There are three interesting points in this case: First, the development of convulsions of a more or less localized character in the course of typhoid fever; second, the fact that there was general thrombosis of the intracranial veins without the sinuses being involved, and, third, the entire absence of any signs of meningitis at the autopsy, although the symptoms during life seemed to indicate the presence of this condition. This last fact is of particular interest in view of the fact worthy of recollection, as already pointed out, that while meningeal symptoms may be well marked in enteric fever, true meningitis is comparatively rare.

When it is remembered that thrombosis of the cerebral sinuses is the usual lesion, and that such an authority as Gowers[1] questions whether primary venous thrombosis ever occurs without sinus thrombosis, and that Macewen,[2] in his classical work on the surgery of the brain and cord, says nothing of marantic primary venous thrombosis, the rarity of this condition is noteworthy. Hirt[3] says it may occur in the veins as well as the sinuses, but Dana,[4] Rosenthal,[5] Gray,[6] and Brill[7] fail to describe it.

We may call attention to the fact that thrombosis of the cerebral sinuses is usually said to be due to an exhausting disease or to infection. In such a case as that just described both these factors were present.

Finally, it is interesting to note that an additional factor in this case still further complicated the clinical diagnosis, namely, a

[1] Diseases of the Nervous System. [2] Ibid.
[3] Ibid, [4] Ibid. [5] Ibid. [6] Ibid.
[7] Article in Dercum's Diseases of the Nervous system.

history that the patient had had two severe head injuries, one twelve years before and one two months before.

A case of evident thrombosis recently occurred in my wards in the person of a student of twenty years. He came under observation on the third day of his illness, and for the next eleven days passed through a marked but moderate attack of typhoid fever. On the fifteenth day of the disease he was suddenly seized with hurried stertorous breathing, rising from 26 to 48 respirations a minute, and his pulse rose from the neighborhood of 116 to 148, and finally to 160. He developed hemiplegia of the right side, unconsciousness, contracted pupils, and the eyeballs were deviated upward. Both pulmonary bases posteriorly filled up rapidly, becoming dull on percussion and developing coarse râles. The skin became cyanotic, and blood-stained mucus was expelled from the mouth by the stormy respirations. He died about ten hours after these symptoms began, with marked retraction of the head and neck. No autopsy was permitted, but from the symptoms I am inclined to regard the condition as due to embolus or thrombus in the lung causing infarction, and in the cerebral vessels causing the paralytic and other nervous symptoms.

Lopriore[1] has reported a case of typhoid fever in a little girl of ten years, in which on the seventeenth day of the disease the patient developed aphasia and great restlessness; the child could understand what was said to it, and there was no paralysis of any of its limbs; the motor aphasia, however, lasted for a period of a month and a half, when the child was gradually taught to speak again. Lopriore believes that this case was due to a microbic embolus, which plugged a branch of the Sylvian artery and thereby influenced the Broca centre.

Convulsions, generalized or local, with coma and delirium may arise from thrombosis of the cerebral sinuses or of the cerebral arteries, but they are very rare from any cause (see hemiplegia article for cases). Murchison only met with them in six cases out of 2960 cases. If due to the lesions named, they indicate a fatal

[1] Gazzetta degli ospedali e delle cliniche, January 5, 1899, p. 25.

termination in the near future. In Osler's case death followed convulsions produced by thrombosis of the branches of the left middle cerebral artery in twelve hours. If they occur in neurotic children or females the outlook is not so gloomy, as they probably do not depend upon an actual lesion in the brain. Thus West has recorded a case in which convulsions developed in the third week of typhoid fever in a child, recurring on two successive days. These were followed by hemiplegia which, however, gradually disappeared in four days. Recovery eventually took place.

During February, 1899, I saw in consultation with Dr. Loux, of Philadelphia, a girl in the third week of typhoid fever with typical hysteria, as shown in the facial expression and in the attitude of her body. Her arms were abducted, her forearms completely flexed at a right angle with the arms, and the hands completely flexed at a right angle with her forearms. This case showed, nevertheless, evidences of profound toxæmia, and died a few days later. When first taken ill she was very hysterical, cried and screamed, and repeatedly asserted if she got typhoid fever she would die.

A possible cause of sudden death during typhoid fever, or in convalescence, is said to be bulbar paralysis. Thus Latil[1] mentions a woman of forty-two years, who suffered from a severe attack of typhoid fever with hyperpyrexia and extreme prostration, but not equally marked nervous symptoms. On the eighteenth day of the attack she suffered from paralysis of the bladder, and on the forty-second day from tetanic contraction of the masseter muscles, with dysphagia and a nasal voice. The respiration became shallow and rapid, the patient seemed greatly oppressed, had an anxious face, and asphyxia so rapidly increased that death occurred in a few hours. It seems to me that there is grave doubt whether this case was not one of peripheral nerve paralysis rather than a central lesion, but that sudden death may occur from a small lesion occurring in the medulla is illustrated

[1] Revue Générale de Clinique et de Thérapeutique, March, 21, 1890.

by a case which has been reported by Libouroux,[1] in which sudden death occurred during the third week of the disease, and an autopsy revealed a small hemorrhage in the floor of the fourth ventricle. There was no other condition which could account for the sudden death of the patient.

Stiffness of certain muscles isolated or in groups is also met with, and may sometimes resemble that seen in lateral sclerosis of the cord.

No less authorities than Hughlings Jackson and Angel Money have stated that knee-jerks are never lost in enteric fever. This is scarcely correct, for I have recently seen a case, not excessively ill, in which they were absent for days at a time as completely as in ataxia or some cases of diabetes.

Restlessness and insomnia, often complained of by the patient, is much more rare than the complaints would indicate. Watchful nurses will report repeatedly and truthfully that such patients sleep the greater part of the night and day, and the lack of sleep is either a delusion or else the few waking moments seem prolonged into hours to the patient. On the other hand, persistent insomnia marked by unnatural quiet, the patient lying with the eyes closed, may lead the careless attendant to report prolonged sleep, when in reality true sleeplessness is present. When insomnia is due to feeble circulation, the use of alcohol stimulation will usually relieve the condition, and morphine may be useful.

We come, then, to the consideration of subsultus tendinum and carphologia. Both of these are signs of grave illness, particularly the latter, but they are neither of them as mortal in their prognostic import as the older authors thought, for patients with these symptoms often get well.

Under the name of "irritation of the brain with depression of temperature," a condition has been described by Liebermeister, which comes on in about the second week of the disease when the symptoms are most violent, and in patients who have had prolonged high temperature. The pupils lose their reaction to light,

[1] Gazette Hebdomadaire de Médecine et de Chirurgie, March 5, 1890.

and symptoms of meningeal irritation develop, or in their place marked mental changes occur, the patient becoming maniacal or deeply melancholic. More noteworthy than all, the temperature suddenly falls almost to normal, and remains there for several days, as long as the symptoms named continue, when it rises again to the points usually met with at that period of the malady, and proceeds as before. Such cases are very rare. In his enormous experience, Liebermeister only met with "eight or ten cases."

Tyson asserts that in cases of typhoid fever in which the patient also suffers from epilepsy, the epileptic attacks are apt to be greatly multiplied in the early periods of the disease; to cease as the disease progresses, and to remain absent till convalescence is established.

Neuritis may come on in typhoid fever in the latter part of the third week or in the fourth week, but it is generally a complication noted during convalescence. (See chapter on convalescence.)

Almost, if not equally rarely, pain in the muscles is developed as the result of a myostitis.

Paralysis arising from typhoid fever usually comes on during the very latest stage of the disease or in convalescence, and is so distinctly an after-symptom, as a rule, that it will be considered under the division in which the late complications and sequelæ are discussed. Rarely, however, as will be pointed out, the loss of power may occur in the middle of the febrile attack.

As an evidence of the rarity of extensive and permanent paralysis of the extremities complicating or following typhoid fever, I may quote the statement of Alexander who, during an experience of ten years and a half in the medical clinical at Breslau, did not meet with a single case of paralysis among 3900 typhoid patients. (Hemiplegia in typhoid fever is discussed later on in the volume.)

The Skin in the Well-developed Stage of the Disease. The rash of typhoid, which usually develops about the seventh or ninth day, is usually characterized by its rose-spot appearance. A delicate pink hyperæmia of the skin is all that it amounts to in many cases, and the rash may be so sparse as only to be found by

the most careful examination of the whole body, when a few spots will reward the search. They are usually found on the belly, the chest, or the back. In other cases the spots are very profuse, being present literally by the hundred. This is rare. During the past year the writer has been impressed by the fact that the rash has been unusually profuse and exceedingly coarse. The individual spots have been not only large and well-defined, but distinctly elevated and maculo-papular to an extraordinary extent. Further, in these cases repeated crops of this roseola have repeatedly appeared as the disease progressed. The rose rash of enteric fever, however, is so typically separated as to its various spots, and there is so little coalescence, that few of the general forms of rose rash resemble it.

In rare instances, however, the rash does coalesce, and then may resemble measles, and in still other cases where its papular form is lacking, this coalescence may render it very much like that of scarlet fever. If the case is enteric fever the abdominal symptoms point to that cause of the rash, while on the other hand, if it is scarlet fever the throat symptoms will point to this malady. In those cases in which marked pharyngeal irritation ushers in typhoid fever, however, the diagnosis may be very difficult. Recently a patient under my care suffered from a mild attack of typhoid fever lasting seventeen days, and ten days later was suddenly seized by a high temperature and general illness. When he came under observation a second time he had a profuse rash over his body; his eyes were injected, and on the mucous membrane of the palate and on the roof of the mouth there was a profuse punctated eruption. The subsequent course of this case showed that he was suffering from a mild typhoid relapse.[1]

The rash of typhoid fever is not a constant symptom, and may appear on the arms and even the hands, instead of on the trunk. In 199 cases under Osler 13.1 per cent. had no rash.

Abnormal eruptions occurring in typhoid fever in children were

[1] For a discussion of the various forms of roseolous rash see the author's Textbook of Practical Diagnosis, fourth edition, 1899.

described as long ago as 1839 by Taupin,[1] who tells us that a uniform erythema resembling scarlet fever may be present, but is not followed by desquamation or œdema. He also says[2] that he has never seen a vesicular rash such as has been described before his time by Prosper Dor.

The other forms of aberrant rash in typhoid fever are usually developed later than the tenth day. They consist in small hemorrhagic exudations or petechiæ. In other cases they may be as large as a silver half-dollar, and do not disappear on pressure. It is as if the rash developed and then hemorrhage took place into the spot.

Another form of skin manifestation in typhoid fever is the tache bleuâtre. They were first described as occurring in typhoid fever in 1837 by Piedagnel. I have been confident that I have seen them in cases which were not infected by lice, but Hewetson[3] speaks as follows in respect to this question:

"There exists a considerable difference of opinion as to the diagnostic value of these spots. Many writers, particularly the English, believe that they are often seen in the early stages of typhoid fever, and have laid some stress upon their presence, although they admit their occasional occurrence with pediculi. Other observers, especially the French, claim that they do not exist unless pediculi, and more particularly the pediculi pubis, are present; that when the spots exist the pediculi or their nits can be found if looked for carefully. Our experience leads us to believe that the latter view is correct, as in the cases of typhoid fever in which the peliomata were present, we were able in each instance to find either the pediculi or their nits. There have been several cases, other than typhoid fever in which these grayish-blue spots were found, but always associated with pediculi. There are at present two cases in the wards, one with catarrhal jaundice and another admitted for chronic bronchitis and emphysema. In

[1] Journal des Connaissances Méd. Chirurgicale, 1839.

[2] This essay is an exhaustive and excellent account of the disease as seen early in this century.

[3] Johns Hopkins Hospital Bulletin, vol. v.

neither case is there any elevation of temperature, but in both there are numerous steel-gray spots scattered over the abdomen, thorax, inner sides of the thighs, and here and there on the arms and legs. In both the pediculi are numerous, particularly over the pubes, and also in the hair over the various sites where the tache bleuâtre are present. In both cases they are quite plentiful in the axillæ, but in neither have they been found on the hairs of the head or face. They do not appear to have caused much irritation; neither patient complained of itching, nor are there marks of much scratching. Indeed, I find that one patient, formerly an Austrian soldier, is quite indignant at the removal of both hair and pediculi. He tells me that they are considered as bringing luck to the bearer, and each sells for from five to ten kreuzers among the soldiers. They have been carefully carried by him for ten years."

Sudamina, due to the retention of sweat drops beneath the epithelial layer of the skin, are met with in cases in which sweating has taken place, during high fever, as a rule. It is claimed by Baradat de Lacaze that sudamina may possess definite prognostic value. In quite an exhaustive paper[1] he concludes that the appearance of sudamina at the beginning of the second week of typhoid fever are of little or no value in fixing the prognosis; but, on the other hand, their appearance again in the second week, or in the period of ambiguity, nearly always indicates the entrance into active convalescence. De Lacaza believes its development at this time means a crisis in the course of the affection.

Urticaria may occur, and there may also be a peculiar mottling of the skin due to local capillary atony.

The so-called tache cérébrale is a red line with white borders, produced in this and other fevers by drawing the finger-nail over the skin of the patient.

Deeper lesions of the skin than those just discussed sometimes complicate typhoid fever. They consist in boils and carbuncles, and are due to infection of the follicles by pyogenic organisms of

[1] Revue de Médicin, 1887, p. 275.

the ordinary forms or by the specific organism of enteric fever. They are usually met with in cases which are severe and characterized by great lowering of the vitality, and are probably more often met with in convalescence than in the acute period of the fever. The writer suffered from a carbuncle on the back, which came on about the twelfth day of an attack and persisted during a relapse and well into the second convalescence.

Bed-sores usually develop only in those cases which are profoundly ill, or are not well nursed, in the sense that they lie in bedding which is soiled by discharges. Since the use of the cold bath or sponging they are rarely met with, because this method of treatment causes the patient to change his posture frequently, keeps him clean, and restores the local circulation in the skin where it is anæmic or congested. The most common seat for this lesion to occur is over the sacrum.

Superficial gangrene of the skin is very rare, but was met with very early in the history of the recognized disease. Thus Taupin[1] mentions a case of sloughing of the thighs, sacral region, knees,

Fig. 16.

Superficial gangrene of the skin occurring in author's wards.

elbows, and of the face, in a child with typhoid fever. The skin became violaceous in appearance and mortified, and this was accompanied by increase in the delirium. In one case under my care at the present time it developed on the inside of the left calf

[1] Journal des Connaissances Méd. Chirurgicale, 1839, No. 7.

of a girl of nineteen years, who had suffered some days before from a series of profuse hemorrhages, for which hypodermoclysis had to be used to save life. None of the areas of injection sloughed, and no injection was given near this spot, which broke down. (See Fig. 16.) Two brown ecchymotic spots formed on the heels where they rested on the bed, but did not slough. The separation of the slough was accompanied by loss of power and sensation in the anterior part of the leg, evidently from peripheral neuritis.[1] (For further discussion of this subject, see the circulation in the developed stage of typhoid fever, and nervous lesions in convalescence.)

Herpes labialis is thought by some to exclude the diagnosis of enteric fever if it be present. Osler reports two cases in which it occurred,[2] and the writer has seen one during the present year.

That herpes occurs quite frequently in some epidemics of typhoid fever is shown by the statement of Zinn,[3] who states that it was met with in 5 per cent. of 190 cases in the hospital at Nuremberg.

A very extraordinary series of cases of gangrene of the skin has been recorded by Stahl, which occurred in soldiers in St. Agnes' Hospital in 1898. He has kindly permitted me to use the following figures. (See Plates I. and II.)

Taupin[4] states that he saw two children die in typhoid fever with severe erythema nodosum, and that sudamina were common in his experience.

Hemorrhagic eruptions may occur in the course of typhoid fever, and, as a rule, they appear in the neighborhood of the joints, when the exudation may be small or quite large.

Nichols[5] reports four cases in which the hemorrhagic diathesis developed on the thirteenth, eighteenth, twenty-eighth, and thirty-

[1] For an interesting paper on infectious disseminated gangrene of the skin, see Caillaud in the Revue Mensuelle des Maladies de l'Enfance, 1897, p. 1.

[2] Johns Hopkins Hospital Reports, 1895, vol. v.

[3] Münchener Med. Wochenschrift.

[4] Journal des Connaissances Méd. Chirurgicale, 1839, No. 7.

[5] Montreal Medical Journal, June, 1896.

PLATE I.

Superficial Gangrene of the Skin Complicating Typhoid Fever.
Stahl's Case.

PLATE II.

Superficial Gangrene of the Skin Complicating Typhoid Fever.
Stahl's Case.

sixth days of typhoid fever. Only one of these cases died. Very rarely the tendency to hemorrhagic leakings may become general and result in hæmoptysis, hæmatemesis, and hemorrhages from the bowels. A case of this character is recorded in the *North Carolina Medical Journal* for September, 1890, in which a child of ten years suffered from this disease. At the end of the fourth week of the disease there was bleeding from the gums, the nose, and blood in the urine. The spots appeared first on the feet and legs, later on the arms, then on the trunk, and, finally, in the conjunctiva.

In other cases hemorrhages other than those just named took place. Thus Hughes and Lévy[1] report a case in which a man, after an ordinary attack of typhoid fever, suffered from a relapse in the sixth week. Abscesses developed in both forearms and in the left arm. When an incision was made into the abscess extravasations of blood into the intramuscular aponeurotic tissues took place, and afterward this was followed by manifestations of acute purpura, as indicated by petechiæ, ecchymoses and severe epistaxis. Recovery took place.

Another abnormality in the typhoid rash has been described by Day.[2] The eruption was on the chest, abdomen, and back, and occurred in irregular dark patches, slightly raised, and disappeared on pressure, though they left some pigmentation after their disappearance. They were not petechiæ. Day asserts that he has met with ten other cases of this character, and further, that in four of them intestinal hemorrhage was foretold by their occurrence in connection with fever, a rapid pulse, and a clear mind.

Eruptive Diseases in the Course of Typhoid Fever. How frequently *scarlet fever* complicates typhoid fever is a difficult fact to decide. Murchison[3] says that in ten years he saw only one case of scarlet fever which contracted typhoid fever, and that ensued on the twenty-sixth day. On the other hand, he cites several cases in which typhoid fever patients suffered later from

[1] Archives de Médecine et de Phar. Militaires, August, 1892.

[2] Dublin Journal of Medical Sciences, March, 1896.

[3] British and Foreign Medico-Chirurgical Review, July 1859, p. 194.

scarlet fever. This was written in 1859. Later still he wrote[1] that in the wards of the London Fever Hospital, in which all fever cases were treated without isolaton, he had seen eight cases in which the eruption of the two diseases existed simultaneously. In one of these the eruption of scarlet fever appeared in the third week of enteric fever, and in the other on the twenty-second day. Indeed, he goes so far in one place[2] as to assert that scarlet fever appears to predispose to typhoid fever.

Sequeira[3] records two cases of typhoid fever complicated by scarlet fever. In one the scarlatinal symptoms developed on the tenth day, and in one five days after the enteric rash. Still more interesting are the cases recorded by Griffiths.[4] Four children, all in the same family, were attacked by both diseases. A boy of eleven years on the sixth day of scarlet fever developed typhoid fever. A girl of thirteen years got scarlet fever three weeks after her brother and enteric fever twelve days later. A girl of three years, who had scarlet fever, suffered from typhoid fever on the eleventh day, and a girl of seven years also on the eleventh day after scarlet fever began. These cases are of special interest in that a nearly simultaneous infection with both fevers must have occurred.

Caiger[5] met with two cases of scarlet fever coincident with typhoid fever, and Payne[6] reports one such case.

Carmichael[7] also has reported the case of a boy of six years, who, after suffering from scarlet fever and going on to the stage of desquamation, continued febrile from oncoming typhoid fever, and Cosgrove[8] records five cases of concurrent scarlet and typhoid fever seen in the Cork Street Hospital. In four of these the incubation stages were concurrent, the scarlet fever being secondary, so that the onset was simultaneous. This same author tells us that instead of increasing the severity of the typhoid the

[1] The Continued Fevers of Great Britain, third edition, p. 586.
[2] Loc. cit., p. 455.
[3] British Medical Journal, 1891, vol. i. p. 849.
[4] Lancet, 1893, vol. ii. p. 1307.
[5] Lancet, 1894, vol. i. p. 1137.
[6] Ibid.
[7] Ibid., p. 246.
[8] British Medical Journal, January 16, 1897, p. 29.

scarlet fever seemed to abort it, though the cases were fairly severely ill. Coombs[1] reports a case in which a boy of eleven years, who had scarlet fever, his family having typhoid fever, was seized on the seventeenth day of his illness by typhoid fever. Gabe[2] reports another case.

The danger of confusing adventitious scarlatiniform rash in typhoid fever with that of scarlet fever was emphasized by Murchison and by Moore[3] and Jenner,[4] and more recently by Bassett.[5] Moore has also seen desquamation take place in this form of rash.[6]

A case of a child of eleven and a half years has been reported by Chrystie,[7] which is of particular interest, because of the fact that measles developed during the attack of typoid fever. Death occurred in convulsions. A similarly constituted attack of typhoid fever and measles is also recorded by Matiegka.[8] The symptoms of enteric fever were well marked on the fourteenth day of the disease, when the eruption of measles appeared over the face and body. A similar case has been reported by Ringer,[9] in a girl of ten years, and Ringwood,[10] records a case in which the child had measles and enteric fever simultaneously, followed by a severe attack of diphtheria, scarlet fever, and chicken-pox, all in the space of seven weeks.

[1] British Medical Journal, February 27, 1897

[2] Loc. cit., April 3, 1897, p. 848.

[3] Accidental Rashes in Typhoid Fever, Transactions Royal Academy of Medicine in Ireland, 1889, vol. vii. p. 10, and Eruptive and Continued Fevers, 1892, p. 371.

[4] Fevers, 1893. [5] British Medical Journal, April 10, 1897.

[6] Loc. cit., January 16, 1897.

[7] University Medical Magazine, December, 1888.

[8] Prager Med. Wochenschrift, September 25, 1889.

[9] London Lancet, June 30, 1889. [10] Loc. cit. July 7, 1889.

CHAPTER IV.

THE COMPLICATIONS OF THE PERIOD OF CONVALESCENCE.

Temperature, Recrudescence, and Relapse. Recrudescence signifies a temporary rise of fever lasting for a few days or a few hours, and is usually due to the ingestion of improper food, to nervous excitement, or, more rarely, it seems to arise from absorption from the intestinal canal of some toxic material which temporarily upsets the balance of heat-production and heat-dissipation. In two instances I have seen full doses of strychnine, given as a circulatory stimulant, produce repeated exacerbations of the normal temperature to the extent of two or three degrees by reason of its irritant effect on the nervous system.

As has already been said, a true relapse cannot be said to have taken place until the physician is assured by another crop of rose rash, enlargement of the spleen, coated tongue, and persistent fever that a second attack is upon the patient. If these distinct signs of another infection are present, then the diagnosis is complete.

Relapses occur in a fairly large percentage of cases, and seem particularly prone to take place in those in whom the primary attack of the malady has been mild. Indeed, the milder the attack, the more likelihood is there of relapse. Further than this, the use of the cold bath in treating the disease increases the frequency of relapse quite distinctly. What the average frequency of this unfortunate occurrence is is difficult to determine, because different epidemics differ greatly in the results they produce, so that in one epidemic relapses will occur with great constancy, and in another almost none will occur. Ord[1] believes that relapses are more frequent in cases with constipation than in those with

[1] Transactions of Association of American Physicians, 1888, vol. iii.

diarrhœa, and that reinfection from within explains their frequency in these instances. In the writer's experience, relapses have been much more common in constipated cases.

In regard to the frequency of relapse it is interesting to note that no less an observer than Murchison places the average percentage at 3 per cent.; Gerhardt, in 4000 cases, 6.3 per cent.; Griesinger puts it at 6 per cent., and Strümpel at 4 to 16 per cent. Berg[1] met with relapse in 12 per cent. of 1626 cases in Curschman's clinic from 1880 to 1892. Eichhorst, in 666 cases in Zurich, found relapses in 4.2 per cent. Zennetz[2] in 384 cases of typhoid fever found 47 relapses, of which 17 were entirely uncomplicated. In the Maidstone epidemic relapses occurred in 16 per cent., and were more common in females than in males. Schmidt[3] found 49 cases of relapse in 561 cases of fever treated in Wagner's clinic from 1882 to 1886, or, if doubtful cases be excluded, 38 relapses, or a percentage of 6.8 per cent., which practically agrees with the percentage obtained by Gerhardt, who in the study of 4000 cases selected from various epidemics, obtained a percentage of 6.3 per cent., while Heman's percentage was 6.5, and Steinthal's, 7.5 per cent. Liebermeister says: "In Basel, before the introduction of this (the bath) treatment, 861 typhoid fever patients gave us 64 relapses, or 7.4 per cent., two of which were fatal; after the introduction of this treatment, 882 typhoid fever patients gave 86 relapses, or 9.8 per cent., ten of which proved fatal. It appears, therefore that the proportion of relapses and the number of deaths are both actually increased under the use of cold water." And discussing the probable bearing of these results, he adds: "At present the probability certainly seems to be in favor of the affirmative of the question (does bathing increase the frequency of relapses?) the more so as it appears that the frequency of relapses is greater in proportion as the antipyretic treatment has been the more systematically employed." Biermer has also found relapses more frequent since the introduction of cold baths. Osler met with 14 cases of relapse

[1] Deutsche Archiv für klin. Med., 1895.

[2] Wiener med. Wochenschrift, September 21, 1894.

[3] Archiv. für klin. Medicin, Band xliii. Heft. 3.

in 160 cases bathed, or 8.7 per cent., but mentions five other cases of doubtful relapses, which raises the percentage; while Shattuck met with 21 in 129 cases, or 16 per cent., and eleven occurred before primary fever ceased. Wilson tells us that it occurred in 11.3 per cent. of his cases, and Osler tells us 8.7 per cent.; Shattuck, 16 per cent.; Immermann, 15 to 18 per cent.; Baumler, 11 per cent.; and Jaccoud, 9 per cent., varying from 7 to 15 per cent. At the Presbyterian Hospital in New York Gilman Thompson found the relapses in 193 bathed cases to be 13.5 per cent., which is 2 per cent. higher than 284 cases treated by all methods during the same time.

There are certain peculiarities in the course of a relapse as to the fever, the circulation, and the other functions which deserve attention. The fever usually rises more abruptly than in the original attack, and then speedily loses its high grade and becomes more moderate. Often it is more irregular and has greater remissions than the primary fever. Whether it be high or low, its course is usually shorter than the original period if that has been of standard length or longer, while if it has been quite short the relapse is not infrequently much longer. Thus in one case recently seen by the writer, the primary fever lasted twelve days, and that of relapse nineteen days. Flint is the only author of note who thinks the relapse is generally worse than the primary attack.

It is interesting to note that in Liebermeister's cases out of 111 cases of simple relapse the fever was longer in duration than in the first attack in 37, shorter in 68, and of the same length in 2. In 29 of the cases the primary attack was mild, and in 82 severe, but the relapses were mild in 47 and severe in 64, and 7 of these died in the relapse.

An important point to determine is the danger of relapse both as to complications and mortality. Here, again, the variation in the severity of the symptoms in relapse is so great that it is almost impossible to reach definite results. It is certain that relapses are not to be regarded lightly, and that they should be recognized with a certain degree of anxiety, even when they appear to be mild in type, because the exhausted state of the patient renders

him more prone to complications and less able to withstand the general toxæmia of the new infection.

This is well shown by the statistics at Basel, when out of 115 relapses hemorrhage from the bowel occurred four times, perforation twice, thrombosis once, pulmonary consolidation nine times, nose-bleed seven times, bed-sores four times, abscesses five times, and petechiæ three times.

To quote Liebermeister again: "If we take the reports of the years 1869, 1870, and 1872 at Basel, we find among 467 typhoid fever patients systematically treated with cold baths, 33 deaths and 55 relapses, 6 of which were fatal; the frequency of relapses, therefore, counting only those patients who had survived the first attack, was in the proportion of 12.5 per cent., as against 9 per cent. before baths were used. The higher rate of mortality among the relapses is of so much greater import, in view of the fact that the relapses, too, were treated antipyretically, which ought rather to have given us a lower death-rate."

The time at which relapses occur is of interest. Usually they take place after the temperature has been normal several days, but in some instances much later than this. More rarely we meet with what has been well called "intercurrent relapse," in which the renewed activity of febrile movement and exacerbations of all the symptoms show that a second infection has been superimposed on the first.

In children relapses are, as a rule, more rarely met with than in adults, although this accident varies greatly in frequency. Among the older writers we find Rilliet and Barthez, who saw only three relapses in 111 patients, while on the other hand, Henoch met with no less than 21 relapses in 137 cases, the relapses taking place after both severe and mild primary attacks, although the mild attacks were most commonly productive of this accident. Taupin, writing in 1839, records two cases of relapse in boys of thirteen and twelve years; both recovered.

As with adults, the relapse usually takes place in children in from three to ten days after primary fever has ceased, although it may occur in the course of the disease in the third week, or

FIG. 17.

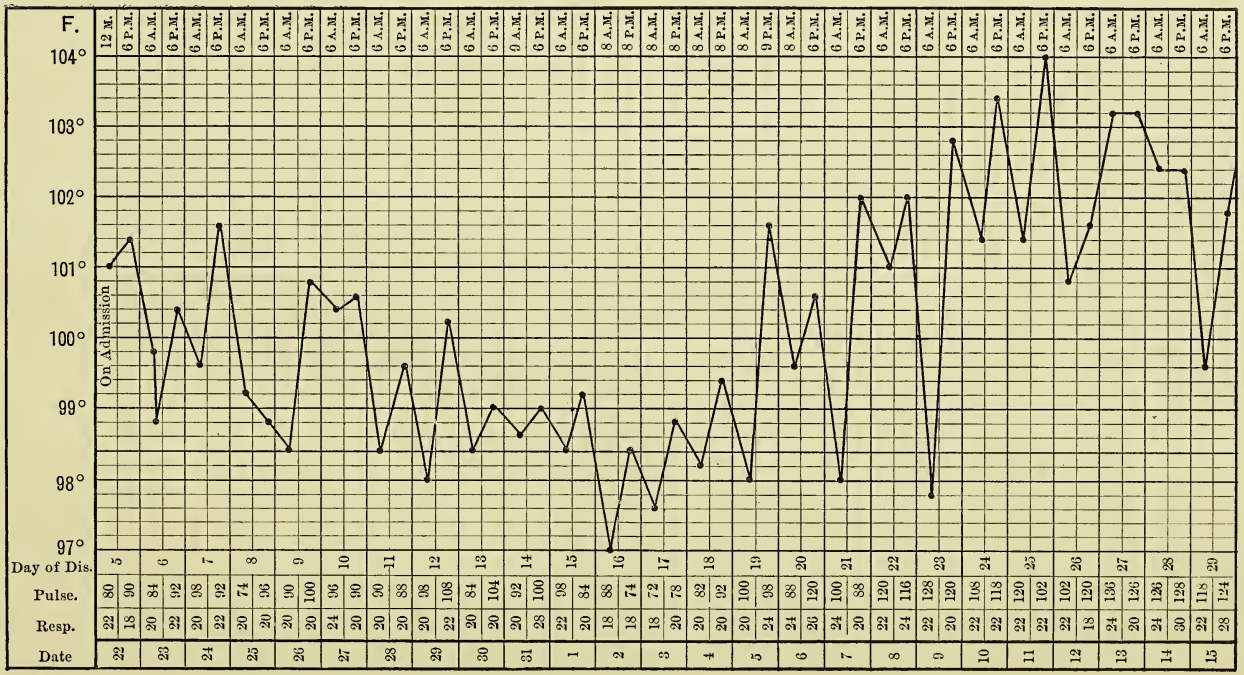


Chart showing mild typhoid fever for twelve days, followed by a relapse lasting nineteen days, this in turn by phlebitis of the left leg, causing a sharp rise on the thirty-eighth day, and this again by a second relapse which ends by crisis on the fiftieth day of illness.

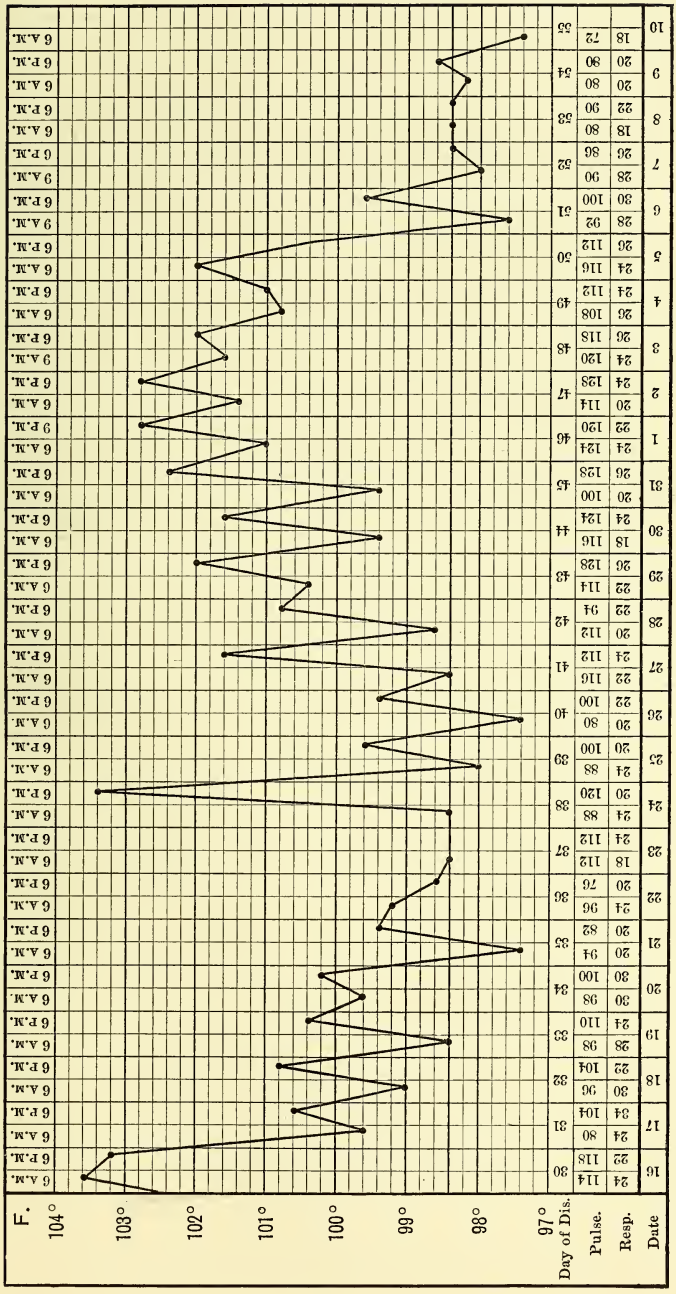
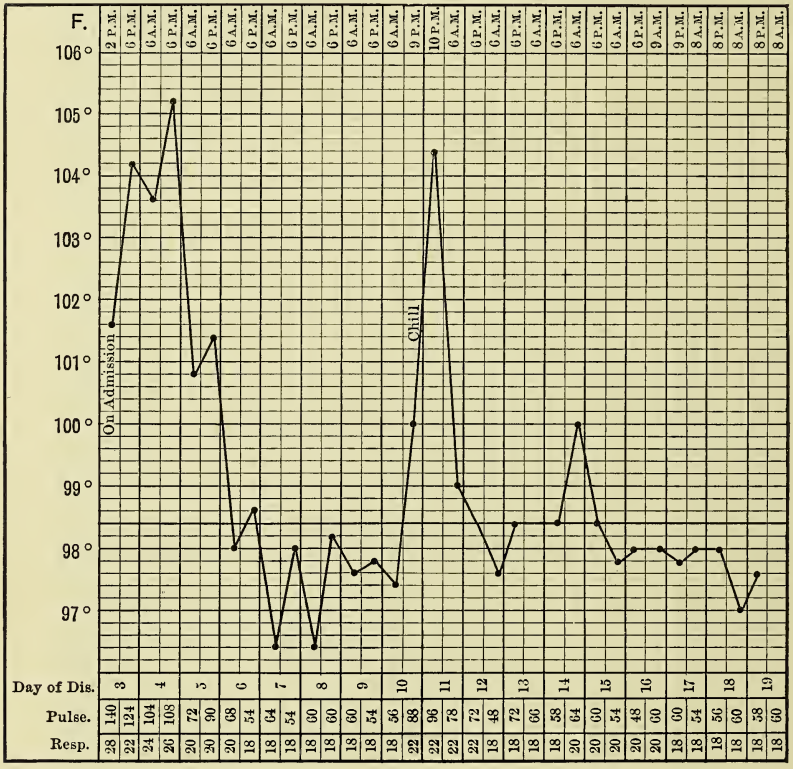


FIG. 17 CONTINUED.

even in the fifth week. Henoch records one instance in which relapse took place in a child eighteen days after apyrexia had been established.

Fig. 18.



Case of typhoid fever in which, according to the patient's story, he had been sick only three days, but in which the disease ended by a rapid fall in lysis, followed by a severe rigor and rise of temperature. The Widal test was positive, and the rash and enlarged spleen were present.

Not only may a patient suffer from a single relapse, but rarely from several relapses. Hutchinson[1] has recorded a case in which three well-marked relapses occurred, and Anders[2] has done so also.

The chart (Fig. 17) shows two relapses.

[1] American System of Medicine, Pepper, vol. i. p. 303.

[2] Medical and Surgical Reporter, July, 1882, p. 66.

Multiple relapses have also been recorded by Johnston.[1] In one case a patient of thirty-nine years had two relapses, and was in the hospital eighty-one days. A second case had two relapses. A third case after a primary attack had two relapses, and the patient was in the hospital 107 days.

A case of typhoid fever is recorded by Carslaw,[2] which suffered from four relapses before ultimate recovery; and I have now under my care a case in his third relapse.

Rigors of considerable severity may occur during convalescence from typhoid fever without possessing any great significance. This is shown in the chart on page 178, and also in that on page 180 (Fig. 18).

Similar cases are recorded by Herringham. Thus he records an instance in which after a mild attack of fever a rigor occurred during the post-febrile period after an enema; another case in which there were several attacks of pyrexia and one rigor during this time, and still a third, in which recurrent collapse appeared during lysis, and rigors in the post-febrile period without any discoverable cause. He believes that ague can be excluded in all of his cases. Herringham also advances the view that in these cases the heat mechanism of the body is so easily upset that very slight causes provoke febrile movement, and in this view the writer concurs. Osler reports two cases of chills without any distinct apparent cause in the later weeks of typhoid fever. In both these cases the chills were followed by hyperpyrexia.

Some years ago Da Costa pointed out that during convalescence from typhoid fever a persistent moderate fever may develop, which is cured by getting the patient out of bed. Shattuck also speaks of such cases. I have had under my care several instances of this character. The getting up ought not to be made till it is evident that the fever is simply a "bed fever" and not a relapse.

Respiratory Affections in the Convalescent Stage of the Disease. Aside from the laryngeal (see earlier and later pages) and other respiratory difficulties met with in the active stage of

[1] Medical Chronicle, May, 1892. [2] London Lancet, July 19, 1891.

the disease, there are no others to be considered at this point save pulmonary abscess, gangrene, and tuberculosis. The latter condition will be discussed in a later chapter dealing with the diseases which ape enteric fever.

Abscess and gangrene of the lung are rare sequences of enteric fever. They arise from one of two causes: either they are due to septic matter which has passed into the bronchial tubes during the stage of stupor, or to septic emboli which first cause consolidation and then tissue break-down. Griesinger met with gangrene of the lung in seven cases out of 118 post-mortems, and Liebermeister found fourteen cases in 230 autopsies of typhoid fever patients.

The question as to whether typhoid fever predisposes the patient to infection by the bacillus of tuberculosis is one of great interest. Cases convalescing from typhoid fever are sometimes met with in which tuberculosis is rapidly developing. In some instances this is due to the fact that the profound depression of the patient's vitality renders him unusually susceptible to any infectious process, but more frequently it is probably due to the fact that the patient has had at some previous time a localized tubercular process which has been walled off from the general system by the usual methods taken by the body for its protection. With the progress of a prolonged exhausting malady vital resistance has decreased, and the local and comparatively harmless process rapidly spreads throughout the body.

In connection with this matter it is interesting to note that Loison and Simonin,[1] in 114 typhoid fever cadavers, found tuberculosis five times, and they point out that typhoid fever may hasten the development of pre-existing tubercular infection. So, too, Sarda and Villard[2] have found the diseases coexisting.

Zinn[3] states that post-mortem examination of the fatal cases in 190 patients revealed the fact that six of them showed

[1] Archives de Médecine et de Pharmacie Militaire, Paris, October, 1893.

[2] Gazette des Hôpitaux, November 30, 1893.

[3] Münchener med. Wochenschrift.

tuberculosis of the lung in association with old foci at the apex.

In cases of typhoid fever which are convalescent the presence of irregular and prolonged febrile movement should raise a suspicion of the presence of pulmonary tuberculosis.

The Circulation in the Later Stages of the Disease and in Convalescence. There are few, if any, diseases which do not have special predilection for the heart muscle or its valves which so gravely interfere with proper circulation as does typhoid fever. The length of the febrile movement and its severity, the gravity of the toxæmia, the wasting of the patient, his inability in certain cases to take sufficient nourishment, and the impaired action of various other vital organs than the heart, all tend to produce weakness in the heart muscle and actual degenerative changes in its nerve-supply and fibres. As long ago as 1875 Hayem[3] made one of his characteristically thorough studies concerning the heart muscle in typhoid fever, in which he showed that a granular parenchymatous degeneration is present in many cases, and that even fatty degeneration may be met with in prolonged severe cases associated with great anæmia. Hyaline changes are not commonly found, but a segmenting myocarditis, in which the intercellular cement substance is softened may be present, although this is, perhaps, a post-mortem change. Many years ago Stokes asserted that the heart muscle of patients dead of enteric fever was so softened that if it were held upside down by its great vessels the muscle would collapse over the hand like a mushroom overspreads its stem. In some cases, on the other hand, the heart seems to escape almost completely.

As it is not the intent of this essay to deal with the microscopical alterations which occur, but rather the objective symptoms of the disease, little further need be said of these changes themselves, except that in this connection the researches of Hoffmann are of

[1] Leçons Clinique sur les Manifestations Cardiaques et le Fièvre Typhoide, Paris, 1875.

interest. He found in an examination of a large number of hearts in typhoid fever patients 56 instances in which the heart muscle was normal or little changed; 39 in which it was slightly granular, the striations still being visible; 46 in which the muscle was granular; 19 in which it was slightly waxy; 1 in which was granular degeneration, and 1 in which it was very waxy.

It is worthy of note that these changes are responsible in a large proportion of cases for the sudden deaths which occur in the convalescent period of the disease, even more commonly than in the course of the disease itself. So frequent is this condition of sudden cardiac failure an accident of convalescence rather than of the febrile attack, that Graves tells us that even if the fever has departed and everything about the patient is favorable, we are not justified in banishing all anxiety or in relaxing vigilance, as a sudden effort on the part of the patient may cause fatal syncope. Instances of this sort have been recorded among the older writers by Bailly, Graves,[1] Jaccoud, and Louis. More recently Dewerve[2] reports that in 48 cases analyzed by him the heart was found softened, pale, and of a "dead-leaf color" in fifteen instances; had undergone fatty or granular degeneration in sixteen instances, and in three others there was proliferative endarteritis of the small vessels of the heart.

Dewerve also found in analyzing these cases that it occurred most frequently in persons between the ages of twenty-two and twenty-five years, probably because this is the age most frequently affected by enteric fever, and that old age and infancy rarely suffered from it. The accident itself is far more common in men than in women, for this writer found it in the proportion of 114 to 26.

It is interesting to note that this condition is not a sequel of severe cases alone, for Dewerve asserts, on the contrary, that it is emphatically a sequel of a moderate form of the fever (*forme moyenne*). Further, violent effort is not necessary to produce it, for

[1] Clinical Medicine.

[2] De la Mort Subite dans le Fièvre Typhoide, Arch. Gén. de Méd., 1887 vol. ii. p. 385.

it has occurred after so slight a movement as extending the arm, by emotion, and may develop without any such cause, the patient being found dead in bed in the posture they were in when asleep. Liebermeister records the case of a woman who ate a hearty dinner after convalescence from a mild illness of typhoid fever. She then rose to go to the closet, fell in a faint and died in ten minutes, and another case of a man who was unable to take the upright posture for many weeks without suffering from nausea, vomiting, collapse, and partial syncope, but who ultimately recovered. The autopsy in the case of the woman revealed no lesions save profound cerebral anæmia.

(For sudden death due to nervous lesions, see chapter on developed stage of the disease, nervous symptoms.)

There are, however, other causes of sudden failure of the heart than myocardial degeneration, namely, embolism or thrombosis of the coronary artery or arteries, heart-clot, thrombosis or embolism of the cavæ or pulmonary veins, and pericarditis with effusion which, pressing on the heart when a change in position is attempted, causes sudden death. In the cases already quoted as having been analyzed by Dewerve (48 cases), there were eight with thrombosis of the coronary arteries. In eight other cases ante-mortem clots were found in the right ventricle. Liebermeister reports one case at Tübingen, in which death occurred as a result of embolism of that branch of the pulmonary artery that goes to the lower lobe of the right lung. In this case the embolus arose from thrombosis of the right crural vein, and was accompanied by extensive hemorrhagic infarction. Clots in the coronary arteries may arise from within the heart cavities from granulations on the endocardium. Further than this, Beaumanoir,[1] Fritz,[2] Vallette,[3] Forgues,[4] Drewitt,[5] and others have met with these formations.

[1] La Progrès Médicale, 1891, vol. ix. p. 364.

[2] Charité Annalen, vol. vi. p. 169.

[3] Contribution a l'Étude de la Gangrene des Membres Pendant la Cours de Fièvre Typhoide, Thèse de Paris, 1890, Ferrand.

[4] Rec. de Mem. de Méd. Militaire, 1880, 3d series, vol. xxxvi. p. 386.

[5] Lancet, 1890, vol. ii. p. 1023.

According to Drewitt, however, these clots are formed in the the heart in the acute period of the disease, and then are dislodged when the circulation increases in tone during convalescence. Viti[1] has found the bacillus of Eberth in the granulations of endocarditis, and, furthermore, has produced these lesions in rabbits by inoculating them with the bacillus, and Vincent[2] has recorded the case of a previously healthy soldier, who died from enteric fever, and in the vegetations of his mitral valves these specific bacilli were found. Girode[3] has made a similar report. Hayem,[4] also, has recorded a case in which endocardial difficulty was recognized in life, and two days later symptoms of plugging of the arteries in both legs ensued. First pulsation ceased in the dorsales pedes, then in the popliteals, and finally in the femorals, and gangrene developed. An embolus was found in the femoral artery, but did not extend below the knee. The autopsy showed ante-mortem cardiac clots, endocarditis, thrombosis of the aorta, and multiple infarctions in the kidney. On the other hand, it must not be forgotten that endocarditis complicating typhoid fever is rare. Osler says he has seen only two cases. Only eleven cases occurred in 2000 cases in Munich. Pericarditis is also very rare (14 in 2000 cases in Munich).

Liebermeister tells us that endocarditis is rare in typhoid fever, and mentions but one case of the severe form, accompanied by a development of excessive warty growths with perforation of two of the semilunar folds, and consequent infarction of the kidneys and spleen, double pleural pneumonia and death. He believes, however, that a mild form of endocarditis without ulceration is more commonly met with.

In other cases embolism of the pulmonary artery results from thrombosis of the femoral vein and causes sudden death. Thus

[1] Atta della Roy. Accad. del Fisiocritia de Siena, 4th series, vol. ii. fasc. 5 and 6, 1890.

[2] Merc. Médicale, February 17, 1892, p. 73.

[3] Comptes Rendu Soc. Biol., 1889, p. 622.

[4] Progrès Médicale, 1875.

Nawercke[1] records a case of this character in which the patient dropped dead when at stool, death coming on in ten minutes, and Bouley[2] reports a case of ascending thrombosis of the femoral veins into the cava and from there into the right auricle.

In other instances an endarteritis may involve the coronary vessels and cause sudden death, if we can rely upon the views of Landouzy and Siredey.[3] These investigators tell us that from the clinical point of view the manifestations of cardio-vascular disease in typhoid fever may present two different aspects. Sometimes the rapid spread of the lesions in the heart and vessels is accompanied by a rapid pulse, with great feebleness of the heart and, perhaps, by its sudden arrest. Sometimes, on the contrary, these changes are developed so slowly and insidiously that death occurs more or less remotely and with variable degrees of cardiopathic change. The symptoms usually met with in the first variety may be classed as those of collapse, with great lack of power in the cardiac muscle. The pulse becomes extremely rapid, small, irregular; the face is livid, the eyes sunken, the voice feeble, and the extremities cold. The temperature may be subnormal. The urine is scanty or suppressed. The respirations are embarrassed, and the lungs are affected by hypostatic congestion. Finally, coma and death come on. This form of collapse may come on as early as the second or third week. The feeble apex beat and rapid pulse indicate a diffuse alteration in the heart muscle, which is usually a fatty degeneration of its fibre (granulo-graisseuse). In cases of sudden death, on the other hand, the lesions are chiefly connected with the walls of the cardiac vessels, the symptoms being in abeyance for the most part till the fatal moment, but dependent upon gradually increasing degenerative processes.

In other cases where the changes are less marked, the patient

[1] Correspondenzblatt für Schweizer Aertze, 1879, 485.

[2] Progrès Médicale, 1880, viii. 998

[3] Contribution à l'Histore de l'Artérite Typhoidique, Rev. de Médicine, 1885. Those interested should also read a paper by Landouzy and Siredey, Étude des Angio-Cardiaques Typhoidiques Leurs Consequences Immediates, Prochaine et Eloignées. Revue de Méd., 1887, p. 804.

does not suffer from severe and alarming symptoms, but instead of these the patient is affected by a disordered circulation and lack of tone in the heart and vessels. The chief signs of these conditions are intermittence of the pulse and a harsh diastolic murmur at the cardiac base.

The cardiac lesions in mild cases may be entirely recovered from so far as symptoms are concerned, but the actual lesions themselves often remain, and Landouzy and Siredey record a case in which a second attack of typhoid fever came on two years after the first, and at the autopsy old and new lesions were found in the myocardium.

As a matter of fact, the cardiac changes of typhoid fever are closely allied to those that are found in cases affected by other infectious diseases of a severe type.

Sudden death in typhoid fever may occur as early as the tenth day. Méry reported such a case to the Société Anatomique in October, 1887. He states that the myocardium did not show any histological changes and that the patient had been treated by the Brand bath. In discussing this case, Cornil spoke of the difficulty of discovering any satisfactory cause for these accidents, and referred to the fact that some persons believed them to be due to changes in the nervous ganglion of the heart—a hypothesis which does not permit of verification.

Pericarditis, as already stated, is rarely due to typhoid infection, although it may complicate its course, being produced by another cause. Thus Hutchinson records a case in which a patient convalescing from enteric fever suffered from erysipelas, then from pleurisy, and finally from pericarditis. Surely this case was due rather to the streptococcus than to the bacillus of Eberth. Liebermeister only saw four cases of pericarditis, and all recovered.

Very rarely sudden death ensues without our being able to find any of the causes given. Déjèrine[1] has recorded two such cases, in which no sign of cardiac degeneration could be found. In such

[1] Comptes Rendus Société Biologie, 1885, p. 769.

instances an embolism of an artery supplying an important vital spot in the medulla may be the cause.

Dieulafoy[1] asserts that in such cases there may be another cause of death, namely, reflex irritation along the vagus from the abdominal cavity, and which, being transmitted along the efferent branches of this nerve, inhibits the heart's action and causes fatal syncope. In other instances he thinks that the respiratory centre is rapidly affected, and that death results. Such reasoning, in view of our knowledge of the functions of the parts of the nervous system just named, seems very hypothetical.

Death due to the causes enumerated may come on more gradually than has been intimated so far. Thus dyspnœa, irregularity of the pulse, a *bruit de souffle*, and, rarely, partial syncope, may begin the end.

Passing from these changes to those met with in the general bloodvessels, we find that marked inflammatory processes often affect these parts in the course of typhoid fever. One of the most important studies made upon this subject is that of Barié,[2] who asserts, as a result of his work, that both the large and small vessels may be affected by inflammation, although the vessels of the lower extremities are the ones most often and most severely affected. Thus in twenty-two out of twenty-four cases this was true. It takes place generally when the patient first leaves his bed and begins to move about. It is just as apt to follow mild as severe attacks, and it occurs in two forms, namely, as an acute obliterating arteritis and as an acute parietal arteritis. He describes the change as follows:

"The first variety is constituted anatomically by an embryonal infiltration of the three coats, and disappearance of the smooth condition of the intima, which becomes uneven and granular. This leads, as a consequence, to the production of a secondary thrombosis, which in course of time becomes a dense gray mass adherent to the parietes of the artery. Very often the inflammation of the

[1] De la Mort Subite dans la Fièvre Typhoide, Paris, 1869.

[2] Contribution à l'Histoire de l'Arterité Aigue Consecutif a la Fièvre Typhoide, Revue de Médicine, 1883, p. 1, and 1884.

artery is accompanied by a certain amount of periarteritis. If the lumen of the affected artery is completely obliterated and the collateral circulation is not quickly established, mortification ensues, and the limb assumes the appearance of dry gangrene. In exceptional cases, in consequence of the simultaneous occurrence of venous thrombosis or of phlebitis, moist gangrene may follow the mummifying variety, or substitute itself for it.

"The principal symptoms of obliterating arteritis are as follows: Acute pain occurring more or less suddenly and seated in the course of the affected artery, sometimes localized in a restricted region, as, for instance, the thigh, calf, or Scarpia's triangle, sometimes occupying the whole length of the limb, and increased by pressure upon assuming the erect position and by the movements of walking; diminution of the fulness and, finally, suppression of the pulsations of the artery; swelling of the limb without œdema or redness; bluish mottling of the skin; sometimes, although rarely, purpura; diminution of the temperature of the limb with or without disturbance of sensibility, such as formication and partial anæsthesia, and, finally, the occurrence in the course of the artery of a hard and tender cord.

"The parietal arteritis is only a variety of the preceding and has, consequently, the same symptoms but in a less degree of development, except, of course, that the hard, painful cord is absent. It is said, however, that the diminution of the pulsations of the artery is occasionally preceded by an exaggeration of their amplitude, and that in a few cases the temperature of the affected limb has been observed to be higher than that of the other.

"It must be borne in mind that some of the symptoms of the obliterating variety may arise from an embolus, but the presence of a valvular murmur and of other signs of disease of the heart, and the suddenness of the seizure, will enable us to recognize without difficulty the cases dependent upon this cause.

"The therapeutic indications in the milder forms are best fulfilled by rest in bed, the application of emollients or soothing ointments to the limb and wrapping it in cotton. In cases in which gangrene has occurred the patient should be supported by

tonics and a liberal diet, and appropriate antiseptic dressing should be applied to the part."

Other reports on this subject have been made by Ferrand,[1] Deschamps,[2] Mettler,[3] Quervain,[4] and Haushalter.[5]

In addition to these interesting researches there are others of even greater interest, as, for example, those of Rattone,[6] who in four cases found the bacillus of Eberth in the arterial walls and obtained pure cultures from this source. The result of this infection and endarteritis is to aid in the formation of thrombi, and these in turn, by plugging of the vessel, cause rapid dry gangrene of the tributary part. (See lesions in the skin.)

The bacilli are supposed to reach the arterial wall by the blood-stream rarely, or by the blood-stream in the vasa-vasorum.

The veins are very much more apt to be affected by thrombus than the arteries, as every one with a large experience with typhoid fever well knows. Haushalter and Vaques have found the bacilli in the walls of these vessels, and Rattone and Haushalter have found them in the thrombi themselves, and also that the endothelium under the clot was destroyed.

As a result of this thrombosis with phlebitis we may have developed phlegmasia alba dolens, but very rarely gangrene because the collateral circulation is more free in the veins.

The clots in the veins may be single or multiple, and may be of very extraordinary size. In de Santi's case[7] a clot extended from the vena cava in the iliac vein down into the femoral vein, and one extraordinary case is recorded by Beaumanoir,[8] in which clots were in the arteries of both legs, in the right ventricle, in the pulmonary artery, in the femoral veins, and in the aorta. Cases of clots reaching from the femoral vein to the vena cava are recorded

[1] Thése de Paris, 1890. [2] Ibid., 1886.

[3] Philadelphia Medical Times, February 19, 1887, p. 339, and New York Medical Journal, March, 1895, p. 289.

[4] Centralblatt für Innere Med., August 17, 1895, p. 793.

[5] Mercredi Médicale, September 20, 1893, p. 453.

[6] Della Arterité Tifosa in Dehu.

[7] Rec. Mem. de Méd. Milit., 1879, series 3, xxxv. 502.

[8] Progrès Méd., 1891, ix. 364.

by Dumontpalier,[1], Sorel,[2] Bouley,[3] and Mackintosh.[4] A case of thrombosis of the iliac veins and the lower part of the ascending vena cava has been reported by Pansini[5] in a case under his care. Œdema, lividity, pain and loss of power in the legs were present. Pansini refers to a statistical article of Vimont, who up to 1890 collected 112 cases from the literature of this character.

A curious case of varicosity of the subcutaneous veins of the trunk and extremities is reported by Mackintosh[6]. The veins involved were the jugular and internal mammary and external pudic, the superficial epigastric, internal saphenous, and superficial circumflex on both sides. It is supposed by the reporter that a thrombus formed at the junction of the iliac veins and inferior vena cava which, becoming engorged, necessitated a collateral circulation. Curiously enough, the patient survived.

Plugging of the veins to a great degree usually results in moist gangrene, as has already been stated.

In regard to the vessels most commonly affected by plugging, we gain very interesting information once more from Keen's classic essay. Out of 90 cases of gangrene, and Keen believes all these cases were due to plugging of vessels, 46 had arterial plugging, of which 8 were bilateral, 19 on the right side, and 19 on the left side. In the veins in 52 cases there was bilateral involvement on both sides in 4 cases; on the right side in 10 cases, and on the left side in 38 cases. Again, in those cases which did not proceed to gangrene, Keen found plugging in the arteries in 15 cases, of which 4 were bilateral, 6 on the right side, and 5 on the left, and in the veins, out of 47 cases, 3 were bilateral, 13 on the right side, and 31 on the left.

These statistics support the earlier ones presented to us by Liebermeister, who met with 31 cases of thrombosis in the veins of the lower extremities among 1743 typhoid fever patients, the

[1] Comptes Rendu Soc. Biol., 1879, 6th series, vol. iv. parts 283.
[2] L'Union Médicale, 1882, p. 521.
[3] Progrès Méd., 1890, viii. 998.
[4] Glasgow Med. Journal, 1892, vol. xxviii. p. 54.
[5] Centralblatt für Innere Med., June 6, 1896.
[6] Glasgow Medical Journal, July, 1893.

majority of whom were men. In his cases also thrombosis usually did not appear until the stage of convalescence, and rarely as early as the third or fourth week. Out of 24 cases, 16 of which were in men and 8 in women, the vessels became plugged eighteen times in the crural vein, five times in the saphenous vein, and once in the popliteal vein. Thrombosis of the crural vein took place in both sides simultaneously twice, four times on the right side, and twelve times on the left. The saphenous vein was affected on the right side once, and on the left side four times, and the thrombosis in the popliteal vein was also left-sided; in other words, this accident occurred five times on the right side and seventeen times on the left. The frequent occurrence of thrombosis in the left crural vein rather than the right, is believed by Liebermeister and by Keen to be due to the slight pressure exercised upon the left common iliac vein by the right common iliac artery, thereby compressing the vein.

Sometimes phlebitis of the calf of the leg develops in place of thrombosis of the femoral vein. Thus Arnaudet[1] records three cases, one in a woman of seventy-five years, another in a woman of fifty years, and the last in a man of thirty-eight years.

The author has recently had under his care a case of this kind occurring in a girl of twenty years, on the left side. In Arnaudet's cases, one was on the left side, the other two on the right.

The rarity with which plugging of a vessel in the upper extremities takes place is remarkable. Thus in 128 cases collected by Keen, only 4 involved the upper extremities alone; 2 involved the arm and leg, and 124 were limited to the legs.

Genito-urinary. Orchitis complicating typhoid fever during the progress of the febrile stage is very rare, but a case was recorded by Marcus[2] in 1812, of suppuration of the scrotum in "stupid nervous fever." Vulpian[3] also states that this complication may follow grave fevers. It is emphatically a symptom of the period of convalescence. Westcott collected for Keen thirty-two cases, but

[1] La Normandie Méd., November 1, 1891.

[2] Archiv für Med. Erfahrungen, Berlin, 1812, i. 546.

[3] Dictionnaire de Méd., 1844, 2d ed., xxix.

Eshner[1] has collected forty-two cases, and has reported one in his own care. The contribution of Ollivier[2] to the study of typhoid orchitis is, however, very exhaustive, and to him belongs the credit of summarizing most of the literature up to 1883. The writer has also met with one case; its history is as follows:

The patient was a physician, twenty-two years old, who was admitted to my wards in the Jefferson Hospital on January 29, 1898, with a history of having been ill for ten days with frontal headache lasting four days, with pains in the lumbar region, and with general debility. There was diarrhœa, with copious watery evacuations from the bowel, and rose spots were present upon the cheek and the abdomen. Nose-bleed occurred after the patient came under observation. Examination of the blood yielded a positive reaction to the Gruber-Widal test. The urine was albuminous on each of three occasions, but tube-casts were not found. The disease pursued an ordinary and uncomplicated course, defervescence taking place on February 22d, and the patient was dismissed well on March 13th. On March 28th he was seized, without obvious cause, with pain and swelling in the left testicle. The pain was agonizing, and the swelling gradually increased until the testicle became many times its normal size. Dr. Horwitz noted the pain as being intense in a degree far beyond that ordinarily encountered in cases of orchitis of gonorrhœal origin. The temperature was as high as 101° between March 31st and April 2d, and it reached 100.3° on April 19th. Otherwise it was practically normal. There was also no urethritis or urethral discharge. A slight effusion into the vaginal tunic took place, but there was no noteworthy involvement of the epididymis. With the application locally of an ice-bag, and of mercurial and belladonna ointments, and the internal administration of opiates, pain was relieved and swelling subsided; but it became evident that an abscess was forming in the left half of the scrotum. Accordingly, an incision was made by Dr. Horwitz on April 23d, and a considerable quantity of pus, together with a portion of the testicle,

[1] Philadelphia Medical Journal, May 21, 1898.

[2] Revue de Médecin, 1883, pp. 829, 960.

was evacuated. The operation was a success, and the patient recovered.

Ollivier[1] believes that orchitis is more common than is generally thought. He reports three cases of his own. Liebermeister[2] met with it three times in 250 cases, and Sorel[3] found it in 3 cases out of 871 typhoid fever cases seen in ten years. Eshner also quotes Betke,[4] who did not meet with it in the records of 1420 cases, and Dopfer,[5] among 927 fatal cases, did not meet it once. Hölscher,[6] in the celebrated 2000 cases in Munich, records a caseous orchitis in but one instance.

As already stated, Eshner's paper is the latest and most exhaustive contribution to this subject, and I have used it freely in these pages. He tells us that in "forty-one cases it occurred during the course of the fever in 12, and during convalescence in 29. It set in in 1 case during the second week of the fever, in 5 during the third week, in 1 during the fourth week, in 1 during the seventh week, in 1 at an unstated period of the disease, in 3 toward the close of defervescence, in 8 at an unstated period of convalescence, in 8 during the first week, in 8 during the second week, in 3 during the third week, in 1 during the fourth week, and in 1 during the sixth week. There was no apparent relation between the severity of the original disease and the occurrence of the complication, which attended mild equally with severe attacks.

"The onset is, as a rule, abrupt, and may take place while the patient is still abed or after he has arisen and is up and about. The first manifestation is often pain referred to the scrotum, though sometimes there is a chill, with elevation of temperature, acceleration of pulse, and headache, so that a recrudescence or a relapse may be suspected. The pain may involve the testicle, the epididymis, and even the spermatic cord, and it may extend into

[1] Revue de Médecin, 1883, iii. 829, 861.

[2] Ziemssen's Handbuch du speciellen Path. und Therap, 1874, ii. B. 2, 189.

[3] Bulletin et Mem. de la Soc. Méd. des Hôp., 1889, lvi. 236.

[4] Deutsche klinic, 1870, 42 and 48.

[5] Münchener med. Wochenschr., 1888, p. 620.

[6] Ibid., January 20, 1891, p. 43.

the loin. Often a sense of weight or heaviness in the testicle is complained of. The scrotum may become red, tense, and œdematous, and effusion may take place into the vaginal tunic of the testicle. Such an effusion was reported in nine of the cases in this collection. The testicle or epididymis or both become swollen and tender, and they may undergo suppuration. Such an outcome was noted in nine of the cases. Micturition is sometimes attended with burning, and the urine may contain the products of catarrhal inflammation, viz., mucus, epithelial cells, and leucocytes. As a rule, however, there is no urethritis and no history of gonorrhœa.

"The testicle is usually attacked first, and in a considerable number of cases alone. In a smaller number the epididymis suffers alone or first. In the majority, however, both organs suffer. Thus, orchitis occurred alone in 13 cases, epididymitis alone in 6, and both orchitis and epididymitis in 20. Both sides seem to be attacked with equal frequency. The right side suffered in 18 cases, the left also in 18, and both sides in 1. The complication lasts, in its acute phase, for about a week or ten days; sometimes its duration is much protracted by suppuration, and often swelling and induration persist for a long time. In several instances the testicle was lost wholly or in part. The complication occurs most commonly at the period of life at which typhoid fever is itself most common. Thus of 26 cases in which the age is stated, 17 occurred between fifteen and twenty-nine years. The age distribution of the cases in which information upon the point is given, is as follows:"

	Cases.
Between 1 and 4 years	1
" 10 " 14 "	2
" 15 " 19 "	4
" 20 " 24 "	9
" 25 " 29 "	4
" 30 " 34 "	2
" 35 " 39 "	2
" 40 " 44 "	1
" 45 " 49 "	1

Of Eshner's cases, 37 are from French sources, 2 from English, 2 from American, and 1 from a Swiss source.

The cause of this complication is not easy to determine. Sometimes it may be due to infection by the bacillus of Eberth, sometimes from pyogenic organisms not peculiar to typhoid fever. Probably the latter are the more common cause. That typhoid bacilli may enter the testicle is proved by the fact that they have been found in the testicle in bodies at autopsy by Chantemesse and Widal without there being any signs of orchitis.

That the bacillus of Eberth may be the cause is also shown by a case of suppurative epididymitis coming on during convalescence, which Strasburger[1] has reported. The patient was a man of twenty-eight years, who suffered from typhoid fever, the diagnosis being confirmed by the Widal test. The disease ran its normal course, and during defervescence the patient suffered from an abscess of the gum, numerous boils, and, finally, from an abscess of the cheek. A microscopical examination of the pus derived from these boils did not reveal any micro-organisms. Three weeks after the defervescence had commenced the patient suffered from violent pain in the right testicle, which became swollen, and an examination revealed an epididymitis, and forty-eight hours later fluctuation appeared, and puncture revealed a small quantity of pus. Two days later the abscess was excised and the patient made a complete recovery. Cultures of the pus revealed the bacillus of Eberth.

Bucquoy has asserted that such attacks are the result of masturbation—a habit, he thinks, frequently practised during convalescence. Hutchinson, on the other hand, thinks that it is due to thrombosis of the spermatic vein.

The orchitis or epididymitis of enteric fever differs from that due to gonorrhœa, in that it is less painful and usually less acutely inflamed. It is, however, rapid in its course to recovery or suppuration, as a rule, and is usually unilateral. Usually the testicle is first affected, and later the epididymis.

The following table is that of Eshner,[2] and gives a complete record of this condition as it exists in literature. Twenty-seven

[1] Münchener medicinische Wochenschrift, January 3, 1899.

[2] Philadelphia Medical Journal, May 21, 1898.

No.	Reporter.	Reference.	Age	Date of appearance.	Duration.	Side affected.	Parts affected.	Result.	Remarks.
1	Chedevergne	Thèse de Paris, 1864. cf. Larquier and Ollivier.	17	10th day of fever.	10 days.	Left.	Testicle and epididymis.	Recovery.	Mild typhoid fever.
2	Bouchut	Traité prat. des Mal. des Nouv.-nés, des, Enf. à la Mam. et des la sec. Enf., 5 ed. Paris, 1867, p. 996.	4	20th day of fever.	.....	Left.	Testicle and epididymis.	Death.	Grave general state; effusion.
3	Hanot	Bull. de la Soc. Anat. de Paris, xlviii. Ann. 1873, 5 Ser., t. viii. p. 589.	18	During convalescence.	28 days.	Right.	Testicle.	Extrusion of testicular tissue; hard nodule in right half of scrotum, one-third size of left testicle.	Urine contained glairy matter; effusion; suppuration.
4	Cervelle	Thèse de Paris, 1874, cf. Larquier.	23	1st or 2d week of convalescence.	1 month.	Left.	Testicle and epididymis.	Nodular induration of tail of epididymis.	Grave typhoid fever; effusion.
5	Cervelle	Ibid.	25	8th day of convalescence.	4½ mos.	Right.	Testicle.	Testicle enlarged; epididymis nodular.	Grave typhoid fever; effusion; suppuration.
6	Widal	Bull. de la Soc. Clin. de Paris, 1877–78, i. p. 142.	...	3d day of convalescence.	8 days.	Left.	Testicle and epididymis.	Nodular induration of epididymis.	Adynamic typhoid fever of moderate severity.
7	Widal	Ibid.	...	10th day of convalescence.	8 days.	.....	Testicle and epididymis.	Nodular induration of epididymis.	Adynamic typhoid fever.
8	Widal	Ibid.	...	20th day of convalescence.	6 days.	Right.	Testicle and epididymis.	Nodular induration of epididymis.	Adynamic typhoid fever, grave type; hematuria.
9	Dieulafoy	Ibid.	...	.....	.....	.....	.....	.....	Suppuration; evacuation of pus.
10	Lereboullet	Ibid.	...	During convalescence.					
11	Lereboullet	Ibid.	...	During convalescence.					
12	Hanot	Arch. gén. de Méd., 1878, vol. ii. p. 595.	21	16th day of fever.	9 days.	Right.	Testicle.	Complete recovery.	No effusion.
13	Hanot	Ibid.	40	During convalescence, 25th day of fever.	8 days.	Right.	Epididymis.	Complete recovery.	Typhoid fever of moderate intensity, testicle a little increased in size.
14	Hanot	Ibid.	32	19th day of fever.	9 days.	Right.	Testicle.	Complete recovery.	Effusion.
15	Sabourin	Bull. de la Soc. Clin. de Paris. 1878–79. ii. p. 229.	...	11th day of convalescence,	6 days.	Right.	Testicle.	Recovery.	Epididymis scarcely enlarged; cyst in head each epididymis.
16	Huchard	Ibid., p. 238.	...	During convalescence.	.....	.....	Testicle.		
17	Laveran	Dict. encycl. des Sci. Méd., 1882, t. xvii. p. 351. See also Ollivier.	...	In course of fever.	.....	.....	.....	Testicle extruded.	Grave typhoid fever; suppuration.

18	Laveran	Ibid.	32	3d day of convalescence.	6 days.	Left.	Epididymis and testicle.	Slight induration of epididymis persisted.	Epididymis principally involved.
19	Laveran	Ibid.	...	Close of defervescence.	13 days.	Left.	Testicle and epididymis.	Epididymis remained indurated.	Epididymis principally involved.
20	Laveran	Ibid.	22	5th day of convalescence.	4 days.	Right.	Testicle and epididymis	Induration of head of epididymis.	Epididymis principally involved.
21	Manley	Lancet, 1882, ii. p. 1065.	...	1st week of convalescence.	10 days.	Left.	Testicle.	Recovery.	Mild attack of typhoid fever.
22	Larquier	Thèse de Paris, 1882, No. 203.	23	3d day of convalescence.	7 days.	Left.	Testicle and epididymis.	Recovery; epididymis enlarged and nodular.	Grave typhoid fever; effusion.
23	Larquier	Ibid.	19	During defervescence.	9 days.	Left.	Testicle and epididymis.	Slight tumefaction of testicle and epididymis.	Typhoid fever of moderate severity.
24	Sadrain	Ibid.	19	13th day of convalescence.	1 week.	Left.	Testicle and epididymis.	Slight enlargement and induration of testicle.	Considerable effusion; symptoms of catarrh of urinary passages; typhoid fever of moderate severity; effusion.
25	Sadrain, also Eloy	Thèse de Paris, 1882. Union Méd., Nov. 18, 1882, p. 818.	47	13th day of convalescence.	1 week.	Right.	Testicle.	Recovery.	Adynamic typhoid fever of moderate severity.
26	Hamilton	Lancet, Dec. 6, 1882, p. 1039.	...	14th day of convalescence.	.....	Right.	Testicle.	Recovery.	
27	Harrison	Ibid., 1883, i. p. 997.	...	20th day of convalescence.	4 weeks	Right.	Testicle and epididymis.	Portion of testicle sloughed.	Suppuration.
28	Ollivier	Rev. de Méd., 1883, iii. pp. 829, 861.	20	15th day of convalescence.	10 days.	Left.	Testicle and epididymis.	Recovery.	Inflammation of right testicle three months before, consecutive to mumps; ataxic typhoid fever.
29	Ollivier	Ibid.	14	20th day of fever; beginning convalescence.	8 days.	Right.	Testicle.	.....	Typhoid fever of moderate severity.
30	Ollivier	Ibid.	26½	11th day of convalescence.	14 days.	Left.	Testicle and epididymis.	....	Typhoid fever of ordinary severity.
31	Tavel	Corr.-Bl. f. Schw. Aerzte, Oct. 1, 1887, p. 590.	35	Close of fever.	5 weeks.	Left.	Testicle and epididymis.	.....	Suppuration in testicle; typhoid-bacilli found in pus.
32	Pein	Thèse de Paris, 1891.	20	4th day of convalescence from relapse.	12 weeks.	Right.	Testicle.	Atrophy of testicle.	Suppuration; typhoid bacilli found in pus.
33	Sorel	Bull. et mém. de la Soc. des Hôp. de Paris, 1889, 3 ser., t. vi. p. 236.	23	4th day of convalescence.	5 days.	Right.	Epididymis.		
34	Sorel	Ibid.	...	Convalescence.	.....	Left.	Epididymis.		
35	Sorel	Ibid.	...	Convalescense.	8 days.	Right.	Testicle.		

No.	Reporter.	Reference.	Age	Date of appearance.	Duration.	Side affected.	Parts affected.	Result.	Remarks.
36	Girode	Arch. gén. de Méd., Jan., 1892. p. 43.	29	23d day of fever.	6 days.	Right.	Epididymis.	Death.	Effusion; suppuration; typhoid-bacilli in pus.
37	Hug	New York Med. Journ., Aug. 3, 1895, p. 151.	11	3d week of fever, defervescence.	Improved rapidly.	Left.	Epididymis.	.....	Typhoid fever of ordinary severity.
38	Hug	Ibid.	28	3d week of fever.	6 weeks.	Right & left.	Epididymis.		
39	Messerer and Gasser	Arch. de Méd. et de Pharm. milit., 1895, t. xxv. p. 228.	23	35th day of fever.	2 weeks.	Left.	Testicle and epididymis.	Slight induration of epididymis.	Grave typhoid fever; typhoid-bacilli found.
40	Salles and Barjon	Gaz. des Hôp., April 16, 1896. p. 463.	...	23d day of convalescence.	.....	Right.	Testicle and epididymis.	.....	Effusion; suppuration; typhoid bacilli found.
41	Berthoud	Arch. de Méd. et de Pharm. milit., July, 1897, p. 1.	20	48th day of fever.	10 days.	Right.	Testicle and epididymis.	Slight induration of tail of epididymis.	Typhoid fever of moderate intensity; effusion.
42	Berthoud	Ibid.	...	During convalescence.	5 weeks.	Left.	Testicle and epididymis.	Epididymis swollen.	Adynamic typhoid fever; urine contained flocculi of mucus and pus.
43	Eshner	.....	39	6th week of convalescence.	1 week.	Left.	Testicle.	Recovery.	Mild typhoid fever.
44	Hare and Horwitz	Personal communication.	22	34th day after defervescence.	5 weeks.	Left.	Testicle.	Portion of testicle sloughed.	Typhoid fever of ordinary intensity; slight effusion; suppuration; only pyogenic cocci in pus.

of these cases had been previously collected by Ollivier, and ten by Westcott for Keen :

While the manuscript of this part of this essay was in the printer's hands the following additional cases were also reported :

Bunts,[1] of Cleveland, Ohio, records the following case of typhoid orchitis : W. C., aged thirty-eight years, private in B Troop, First Ohio Volunteer Cavalry, was taken sick with typhoid and admitted to the Regimental Hospital, at Lakeland, Fla., August 12, 1898. He had never previously had an attack of typhoid fever. The fever pursued a moderately severe course, and on September 15th he was sent home to Ohio on sick furlough. On his arrival at home he was practically confined to his bed until October 12th. On October 13th he was suddenly attacked by a severe chill and great prostration. He was immediately sent to the hospital, and was confined to his bed for several weeks with what was diagnosticated by his attending physician as a relapse of the typhoid. On September 29th, fourteen days after his discharge from the hospital at Lakeland he noticed a swelling in the left testicle. The pain was moderately severe, increasing as the swelling increased, and at the time of his admission to the hospital in Cleveland he suffered considerable pain, which, however, was relieved by rest, elevation, and hot applications. The relapse was severe and his condition most critical. However, convalescence eventually ensued, but the orchitis remained. No history of gonorrhœal or syphilitic infection could be elicited and the orchitis was diagnosticated to be a sequel of typhoid fever.

Strapping was resorted to in the hopes of reducing the swelling, but was abandoned at the end of a week, no improvement having taken place. After this symptoms of softening and breaking down of the organ became manifest, and it was decided to remove the testicle. This was done November 16, 1898, the only item of interest connected with the operation being that the pulse-rate during the entire operation ranged from 160 to 180, ether being the anæsthetic given. The testicle was found entirely disorgan-

[1] Medical News, March 25, 1899.

ized and a considerable amount of pus was also present. The specimen was sent to the Pathological Laboratory of the Western Reserve Medical College and examined by Dr. Howard, who reported that it contained a practically pure culture of the typhoid bacillus.

Beckell[1] reports the following case of epididymitis complicating typhoid fever: M. G., aged forty years, ran a rather severe course of typhoid fever; was much prostrated. During the fourth week of the disease the left epididymis became greatly swollen, and suppuration resulted. This condition did not cause much constitutional disturbance. A free incision and gauze packing soon effected a cure.

Alimentary Tract and Associated Organs in Late Stages and in Convalescence. The affections of the alimentary canal after typhoid fever are not, as a rule, of very great importance nor of great frequency. In the majority of instances they consist in more or less severe signs of indigestion due to three factors, namely, the inordinate appetite of a patient convalescing from typhoid fever, which often leads him to overload his stomach, his inability to deal with ordinary amounts of food is impaired by his generally feeble state, and, finally, the disordered condition of the bowels, as represented by the states of diarrhœa or constipation, may be prime factors in interfering with the proper digestion of food.

Obstinate and persistent constipation is the condition of the intestine most commonly met with, and it varies from a moderate form readily relieved by proper diet and drugs to a condition in which the fecal mass must be dug out of the rectum with a spoon. This condition is due to two chief causes. In the first place the tissues are so dried out by the fever, so to speak, that they eagerly absorb from the alimentary canal all the liquid they can to restore their normal moisture; and, secondly, the prolonged use of a diet leaving but little residue, and lack of exercise is a causative factor of intestinal atony, even if the ulceration and catarrhal state of

[1] Southern California Practitioner, March, 1899.

the mucous membrane of the bowel in the disease are not considered.

Diarrhœa may also be a factor which delays the patient's rapid return to health, and it arises from the use of improper food, from catarrh of the bowels, or from the presence of unhealed ulcers in the colon, or even in the small intestine. This condition of faulty healing of the ulcers in the bowel may be a serious factor in the patient's case. Rarely serpiginous ulceration of the mucous membrane of the bowel is present, and this results in a persistent diarrhœa of a dysenteric type with, it may be, loss of blood. This condition has been described by Jaccoud in France, and by George B. Wood in America, and by many other clinicians since his time.

In other cases perforation of the bowel may take place with death therefrom long after the fever has departed. Thus Morin[1] has recorded a case in which perforation occurred as late as the one hundred and tenth day. Sometimes these ulcers, by affording foci for septic infection, cause the maintenance of a low grade of fever for many weeks. They are not true typhoid ulcers, but the result of profound necrosis of the intestinal mucous membrane resulting from advanced intestinal catarrh and debility.

Under the name of diphtheria of the intestinal mucous membrane, Liebermeister has described a condition in which the bowel is affected by diphtheroid sloughs. Very rarely, if ever, are these sloughs truly diphtheritic. The ulceration underlying them may be severe enough, however, to result in perforation of the bowel, as already pointed out.

Gangrene of the bowel in distinction from ulceration and local necrosis is still more rare. It is probably due almost always, if not always, to thrombosis or embolism of the mesenteric vessels, and in Hoffmann's 250 cases at autopsy this lesion was found no less than nine times. In six of these it affected the ileum, in two the vermiform appendix, and in one the sigmoid flexure. Those cases in which there is gangrene of the appendix are probably due

[1] Des Perforations Intestinal dans le Cours de la Fièvre Typhoide, Paris, 1869.

to appendicitis, produced by direct infection by the bacillus of Eberth or by the bacillus coli communis. (See earlier chapter.)

Peritonitis arising from infection from the ulcers in the bowel wall or from perforation may also arise in this period of the disease. Tschudnowsky[1] records a case of this character in which, after typhoid fever, perforation occurred with the escape of gas into the peritoneal cavity. Auscultation in this case revealed an exquisite amphoric murmur on inspiration due, it was thought, to the escape of gas through the opening in the gut.

Cicatricial contraction of the bowel due to the healing of the ulcers is an exceedingly rare condition, which is a curious fact, when we consider how severe the ulcerative process may be. Young[2] has recorded a case, however, in which the lower twenty-five inches of the ileum were so greatly contracted that the first joint of the thumb could not be inserted into the bowel. In this case, too, about two inches above the ileo-cæcal valve there was constriction, almost to the point of occlusion, and a similar narrowing existed at the upper end of the contracted portion of the bowel. Above this upper constriction the small bowel was so dilated that it resembled a stomach. The patient died as the result of a fall from a horse long after the typhoid attack.

Concerning the more infrequent complications affecting the alimentary tract at this period, we find a number of interesting facts. Noma has been recorded in a few cases, notably by Freymuth and Petruschky,[3] who report a case of noma of the cheek in a case of typhoid fever in which virulent diphtheria bacilli were isolated from the gangrenous tissue, and in which healing followed the use of antitoxic serum. Keen collected nine cases in his Toner Lecture in 1876, although some of these were rather those of cancrum oris than true noma, and Hall has reported to Keen a case which, as Keen says, if not one of noma was at least akin to it. The patient died of hemorrhage from the area involved on the thirty-eighth day of the general malady. So, too, Littlejohn[4] has re-

[1] Berliner klin. Wochenschrift, 1869, Nos. 20, 21.

[2] Medical Press and Circular, 1886, xlvi. p. 471.

[3] Deutsche med. Wochenschrift, 1898, No. 15, p. 232, and No. 38, p. 500.

[4] British Medical Journal, April 30, 1893.

corded two fatal cases of noma following typhoid fever. In one of these both cheeks sloughed; in the other there was not only sloughing of one cheek, but gangrene of the skin of the hip.

Aphthous inflammations of the mouth may be present in rare cases, and is usually seen only in patients who are in crowded wards or barracks, in which careful attention cannot be paid to individual cases.

Glossitis may occur in typhoid fever, but is very rare. Osler has recorded a case which developed glossitis ten days after his temperature was normal, but recovery ensued in a few days.

Alveolar abscess may also occur, and Liebermeister records a case in which there was emphysema of the cheek of the afflicted side.

Franklin[1] has reported a case in which gangrene began in the upper gum and caused in five days necrosis of the superior maxilla.

A case of gangrene of the mouth and partial necrosis of the superior maxillary bone has been reported by Winkouroff,[2] as occurring in a little girl six years old. The left cheek was observed to be swollen on the first day of the illness; on the third day a black spot made its appearance in the back of the mouth; on the seventh day the eschar suppurated and perforatiou of the cheek occurred. The most noteworthy fact in this case is that of recovery.

Induration followed by softening and perforation of the cheek, and finally by death, has been reported by Donald[3] as having occurred in two sisters during the course of typhoid fever. In both cases the right cheek was affected. I have under my care at present a woman convalescing from a most grave attack of typhoid fever, with an abscess forming in the wall of the right cheek which is not connected with the parotid gland or Steno's duct.

Keim[4] has reported a fatal case of typhoid fever in a boy of

[1] Quoted by Hutinel.

[2] Bulletin de la Société Anatomique, December, 1887.

[3] London Lancet, February 20, 1893.

[4] Lehigh Valley Medical Magazine, October, 1891.

nine years, in which gangrene of the left cheek occurred during convalescence. Two other cases are reported in the same journal.

Another case has been reported by Clark,[1] in which a man of twenty-eight years suffered on the thirtieth day of typhoid fever, with bulging of the right cheek, followed by closure of the right eye and great swelling of the lids, and on the thirty-third day the left eyelids became involved, and on the thirty-fifth day large non-glandular swellings appeared at the angles of the lower jaw. The right upper eyelid sloughed away, and the patient died of exhaustion on the thirty-seventh day of the illness. It is thought that the local condition was the result of a general infection.

Sloughing of the face in a child of twelve years, ending fatally, is reported by Ewens.[2] In this case the sloughing really followed an attack of measles and mumps which occurred during convalescence in typhoid fever.

Gangrene of the tongue has been reported once by Gaston David,[3] while Freudenberger[4] has seen it involve the uvula. Spillmann[5] met with gangrene of the lips with final septicæmia due to a secondary staphylococcus infection, which destroyed life.

Liebermeister records one case of melanotic softening of the œsophagus after typhoid fever.

Œsophageal ulceration[6] may lead in some cases to stricture. A case has been reported by Packard, and one by Mitchell which occurred in Osler's wards.

A case of ulcer of the œsophagus has been recently reported by Riesman to the Pathological Society of Philadelphia, March 9, 1899. (Fig. 19.)

In regard to lesions coming on at the other end of the alimentary canal after enteric fever we find a case of gangrene of

[1] London Lancet, April 9, 1893.

[2] London Lancet, August 4, 1889.

[3] Quelques Considerations sur la Gangrene Typhoide. Thése de Paris, 1887.

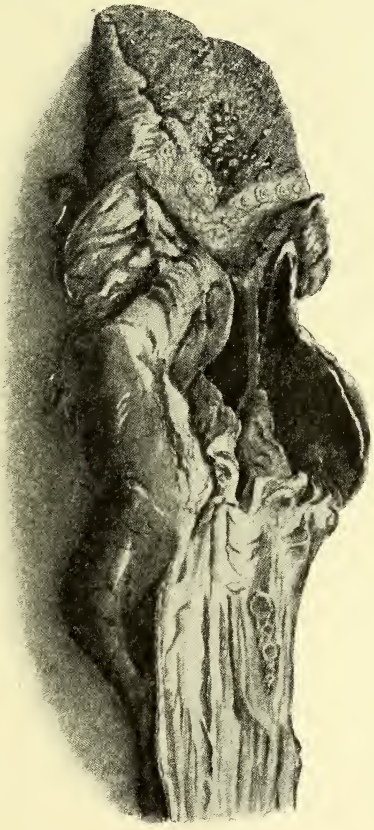
[4] Aertzliche Intelligenzblatt, 1880, xxvii. 7.

[5] Merc. Medicale, 1895, No. 13, 145.

[6] A valuable paper, by Russell, on œsophageal ulceration in general is to be found in the Scottish Medical and Surgical Journal for April, 1899.

the anus reported to Keen by Betz, of Oakville, Pa., the condition arising in all probability from general thrombosis of the hemorrhoidal arteries. This patient was a boy of ten years, who at the end of the fifth week complained of irritation about the anus, the parts being found slightly discolored. Within twelve hours the

FIG. 19.



Riesman's case of ulcer of the œsophagus in typhoid fever. (Case reported in the Philadelphia Pathological Society's Transactions, March, 1899.)

tissues of the ischio-rectal fossa sloughed out and the rectum was found to be gangrenous. It speedily separated, leaving a large opening. Curiously enough, absolute recovery took place, the evacuations being finally perfectly controlled.

Cases of gangrene of the perineum and anus may occur from extension of the process from the vulva in women. Keen gives interesting facts concerning these cases which are not medical conditions and, therefore, need not be discussed at this time.

Passing on to the lesions found in the organs associated with the alimentary canal, we find that inflammation of the parotid gland is an unusual complication of typhoid fever, and is due to extension of infection from a foul mouth through Steno's duct. In rare instances, however, the parotitis is due to true typhoid infection. Thus Janowski[1] records a case of a man of twenty years who died in the "second or third month" of the fever. The bacillus of Eberth was found to be the infecting organism in the gland. In another case,[2] both the bacillus of Eberth and the staphylococcus were found to be present. Sometimes the inflammatory process goes no further than swelling and hyperæmia; in others suppuration develops, and when it does the destruction of tissue is usually grave, not only in the gland but in nearby tissues as well. Curiously enough, the other salivary glands are almost never affected. Not only may the local necrosis be dangerous in itself, but if the pus is not given free vent it is apt to burrow down between the tissues of the neck and cause septicæmia or pyæmia by infecting the great vessels and lymphatics. Facial palsy may result either from destruction of the facial nerve, by its section in incising the abscess, or by reason of the pressure exercised upon the nerve as it passes through the stylomastoid foramen, the neighboring bony tissues being involved. In regard to the frequency of this condition, we find that Hoffmann met with suppurative parotitis in 16 cases out of 1600 patients, and that 7 of these died. Ordinary parotitis occurred in 3 cases. In 15 cases the attack was limited to one side, 9 times in the right and 6 times in the left. Keen collected 26 cases in his Toner Lecture of 1876, and 50 more in his recent essay. Thirty per cent. of these died, and 20 of the 28 cases in

[1] Centralblatt Bacteriol. und Parasit., 1895, xvii. 685.

[2] Lehman. Centralblatt für klin. Med., August, 1891, 649.

which the sex was named were males. Twenty-nine of his cases suppurated and only 5 did not. In 12 the trouble was bilateral, and 7 of these suppurated on both sides.

Parotitis is a lesion of the third or fourth week, and is of evil omen, since it shows degenerative changes in other important glands.

Osler has recorded a case in which a right parotid abscess complicated typhoid fever in a man who was ill in September, 1890. In January, 1896, when Osler saw him, he had profuse sweating over the right side of the face and temple on eating, this condition having lasted more than five years. There was no facial anæsthesia or paralysis.

The liver may become affected by various conditions in convalescence. Of these we find, as most important, abscess, cholangitis, and cholecystitis.

Here, again, the exhaustive monograph of Keen may be referred to as presenting many of the facts we have concerning this organ. Abscess of the liver is seldom met with, for Keen found only twenty-one cases in literature. Solitary abscess is due to the bacillus coli communis, to the staphylococcus, or to the bacillus of Eberth, and is very rare. Osler has not met with it once, and in the Munich 2000 autopsies[1] it was only met with in twelve cases, while Dopfer, in 927 cases, found abscess in only ten of them. Out of the twenty-one cases of solitary abscess no less than nineteen cases died.

When there are septic foci elsewhere the abscess is usually secondary and multiple. Louis has recorded a case of hepatic abscess associated with parotid suppuration, and Chvostek one consecutive to perichondritis of the larynx. Delaire[2] has reported an instance in which an hepatic abscess ruptured into a bronchus; the abscess was incised and recovery occurred.

Lannois reports the following case, which occurred in the Hôpitaux Militaire de la Charité in 1881 : A man of twenty-two years,

[1] Hölscher. Münchener med. Wochenschrift, 1891, Nos. 3 and 4.

[2] Gazette des Hôpitaux, 1869.

after several days of malaise, presented all the signs of adynamic enteric fever. In the third week he became intensely jaundiced, "fairly black;" the liver was enlarged; there was active delirium and intense pulmonary congestion. Eleven days after the onset of the jaundice a small superficial abscess appeared on the back of the left hand and on the right side of the face. The autopsy revealed the ordinary lesions of typhoid fever, congestion of the lungs, and an enormous hepatic abscess of 3000 grammes (3 quarts). The pus was yellow and greasy, and the gall-bladder was distended with clear liquid and muco-pus. The other case recorded by Lannois[1] is somewhat different from this, in that the symptoms of abscess developed after the fever had ceased. On the third day of apyrexia the patient, who was a young man of twenty-eight years, was seized by a violent chill, followed by high fever and at the same time by signs of "pleuro-pulmonary" disease at both bases, but chiefly at the right base. Ten days later the belly was tympanitic, and there was tenderness in the hypochondrium of the right side. Rapid emaciation ensued; the pulse became feeble, and the patient oppressed. Sharp pain was suffered in the epigastrium. There was no œdema or albuminuria. The autopsy revealed old lesions of enteric fever, and in the vena porta a large thrombus which extended into all the neighboring branches. Ten large abscesses were found in the lower part of the right lobe of the liver. They varied in size from a mandarin orange to that of an egg. The pus was creamy yellow. Pleural effusion was present.

Multiple abscesses of the liver have been recorded by Romberg[2] after a severe attack of typhoid fever complicated by hemorrhage and followed by jaundice; death occurred. Miliary abscesses were scattered through the liver in large numbers, and there was suppuration of the mesenteric glands with thrombosis of the portal vein and its branches.

[1] Revue de Médecin, 1895, p. 913. Pyléphlébite et Abscès de Foie Consécutif à la Fièvre Typhoide.

[2] Berliner klin. Wochenschrift, March 3, 1891.

Another case of multiple hepatic abscess complicating convalescence in typhoid fever, has very recently been reported by Herman,[1] of Memphis. The patient was a man of twenty-six years, a fireman by occupation, who on the thirty-third day of his illness was seized with a chill and severe lancinating pain in his right side, followed by a rise in temperature and marked tenderness in the liver, but no physical signs of pulmonary trouble. Three days later the patient suffered from rigors and sweats. An aspirator revealed pus, and upon the ninth rib being resected, six ounces of chocolate-colored pus escaped. Later, another rise in temperature with sweats indicated the presence of further pus-formation, and exploration revealed additional abscesses which discharged pus when their walls were broken down by the finger of the operator. This happened a third time, and in each instance when the pus was evacuated temporary improvement took place, but the patient finally died from exhaustion.

Suppurative pylephlebitis is another rare state and may cause hepatic abscess. It arises usually as the result of thrombosis of the vena porta. Schultz found, in studying the statistics of 3686 cases of typhoid fever in Hamburg, that 302 deaths occurred, but no instance of this condition was met with. Buckling[2] found this lesion in two cases. Romberg,[3] who studied 677 cases with 88 deaths, found one instance, although he refers to four more. Staphylococci were found in the thrombi and in the pus. Osler[4] saw one case in which multiple abscess of the mesentery was present, and the portal vein outside of the liver was an elongated abscess. So, too, Lannois[5] records a case of thrombosis of the portal, splenic, and inferior mesenteric veins, with multiple hepatic abscesses. In this case the specific bacillus was found in the pus. Klebs[6] has recorded a case of suppurative cholangitis in which the bile passages were dilated into large abscess cavities.

Cholecystitis, unlike the hepatic complications of typhoid fever

[1] Memphis Lancet, 1899. [2] Fälle von Leber Abscesse, Berlin, 1868.
[3] Berlin. klin. Wochenschrift, 1890, 192.
[4] Trans. Assoc. American Physicians, 1897, 382.
[5] Revue de Médicin, 1895, 909. [6] Handbuch der Pathol. Anatomie.

just reviewed is as common as they are rare. Thus Louis[1] states that changes in the gall-bladder are much more frequent in the course of typhoid fever than in any other disease.

Westcott collected for Keen 74 cases of true typhoid infection of the gall-bladder, of which 30 were operated on. Notwithstanding this comparatively small number in which the bacillus was proved to be the cause of the affection, literature teems with cases in which typhoid cholecystitis was present. Aside from Louis's description of it, we find Andral and Grisolle writing on it as long ago as 1835, and later Rokitansky,[2] Frerichs,[3] and Budd[4] recorded such cases. In America as long ago as 1846 Ayres[5] reported the case of a young physician so affected, who died of peritonitis, and Murchison[6] tells us that "fatal peritonitis may result from ulceration of the gall-bladder proceeding to perforation."

Among those who have written on this very important theme still more recently we may name Chiari,[7] Dupré,[8] Courvoisier,[9] and Hagenmuller,[10] the latter collecting eighteen cases.

It was not until 1890 that Gilbert and Girode[11] proved that suppurative cholecystitis arose from typhoid infection.

It has also been proved that the bacillus of Eberth may remain for many months in the gall-bladder before it produces grave disorders. Thus Dupré[12] records a case in which, at a cholecystotomy, the bacilli were found in the gall-bladder six months

[1] Typhoid Fever, Trans. Bigelow, 1836, vol. i. 269.

[2] Manual of Path. Anat. Sydenham translation, vol. ii. p. 160.

[3] Disease of Liver, vol. ii. p. 454. Sydenham translation.

[4] Diseases of Liver, 3d American ed., Philadelphia, 1857.

[5] New York Journal of Medicine, 1846, vol. vii. p. 315.

[6] Continued Fevers of Great Britain, pp. 566 and 634.

[7] Ueber Cholecystitis Typhosa. Prager med. Wochenschrift, 1893, No. 22.

[8] Les Infections Biliares. Thése de Paris, 1891.

[9] Casuistisch Statistische Beiträge zür Pathologie und Chirurgie der Galburwega, Leipzig, 1890, pp. 76 and 94.

[10] Cholecystitis Typhosa. Thése de Paris, 1876.

[11] Mem. de la Société de Biol., 1890; La Semaine Méd., 1890, No. 58, and Mem. de la Société de Biol., 1893, p. 986.

[12] Les Infections Biliares. Thése de Paris, 1891.

after the fever ceased, and Chantemesse[1] records an instance eight months after the fever, while von Dungen[2] recites one remarkable instance of cholecystitis fourteen and a half years after the fever. *In the pus of this case the Eberth bacillus was found.*

The American writers on this topic have been chiefly Mason,[3] of Boston, and Osler.[4] Mason tells us that the records of the Boston City Hospital show only three cases of this character other than his own. Two of these died. His own case recovered after the gall-bladder had been tapped.

In many of these cases gallstones have been found present, and these probably aid in opening the way for infection, but Bernheim[5] and Chantemesse[6] advance the view that the infection aids in forming the stones. So firm are the French in the belief that this view holds true that they called this form of the disease "hepatic typhoid,"[7] and Dufourt[8] has recorded nineteen cases of biliary lithiasis which had their first attacks after enteric fever and all of them within ten months of the fever. Gilbert and Fournier[9] divide cholelithiasis into two groups: those which are the more numerous, being due to the colon bacillus, and the less frequent form, due to the bacillus of typhoid fever.

A case has been recorded by Anderson[10] in a man of sixty-seven years, who, two months after typhoid fever, was seized with intense pain in the right hypochondrium, followed by death in ten days. The autopsy revealed peritonitis and perforation of the gall-bladder due to the bacillus of Eberth or the bacillus coli communis. Alexieef[11] also reports a case in which a child of five years suffered from a pear-shaped tumor in the hepatic area, and great pain. Operation revealed suppurative cholecystitis, with the

[1] Traité de Méd, i. 764.
[2] Münchener med. Wochenschrift, 1897, No. 26, 699.
[3] Transactions Assoc. American Phys., 1897, xii. p. 23.
[4] Ibid., p. 378. [5] Dict. Encyclo. de Dechambre, 1889. Entire art.
[6] Quoted by Dupré, loc. cit.
[7] Landouzy. Gaz. des Hôpitaux, 1883, 841, or Mathieu, Rev. de Méd., 1886.
[8] Revue de Méd., Paris, 1893, p. 247.
[9] Compte-rendus Soc. Biol., March 5, 1897, p. 936.
[10] Canada Lancet, 1896. [11] Quoted by Osler, ibid.

typhoid bacillus in the pus; recovery occurred. Hawkins[1] reports a case of this character in which after death there were found typhoid lesions, and Osler[2] records four cases, three of which recovered and 1 died. He also records two cases of hepatic colic. one of which followed enteric fever, and one which had typhoid bacilli in the gall-bladder without having had typhoid fever. Cushing[3] tells us that a prior history of typhoid fever is often met with in gallstone cases in Halsted's clinic at Baltimore; and that it occurs in the proportion of 10 in 31 cases. Hektoen[4] also tells us that he has recently seen a case in which the pus from a suppurative lithiasis of the gall-bladder gave the Widal reaction. This patient had typhoid fever six years before. Cushing has also reported in the *Johns Hopkins Hospital Bulletin* for May, 1898, a case in which cholecystotomy was performed for a cholecystitis, in which the typhoid bacillus was found, although there was no history of typhoid fever. The blood in Cushing's case also gave the typhoid reaction. Cushing suggests that the typhoid bacilli enter the gall-bladder, as they have been shown to do by Futterer,[5] and remain alive a long time, during which an agglutinative reaction takes place, forming a clump about which the material for the formation of a stone clusters.

Finally, it is interesting to note that in the mind of no less a pathologist than Chiari, it is held that relapses in typhoid fever may ensue from the gall-bladder infection.

The diagnosis of gall-bladder infection rests on the following points: Tenderness on pressure a little above and to the right of the umbilicus. There is pain in the gall-bladder and under the scapula, and often a pear-shaped mass can be detected in the anterior hypochondrium. This may fluctuate. If perforation occurs peritonitis speedily develops. As Mason well says, in diagnosis we must exclude impacted feces, hydronephrosis, cyst, displaced kidney, and appendicitis, and when rupture of the gall-bladder

[1] Lancet, January 30, 1897.
[2] Ibid.
[3] Johns Hopkins Hospital Bulletin, May, 1898, No. 86.
[4] Progressive Medicine, March, 1899.
[5] Münchener med Wochenschrift, 1888, No. 19.

has occurred, intestinal perforation. Leucocytosis would be indicative of acute cholecystitis and appendicitis.

The prognosis of cholecystitis is grave. Only one-quarter of the cases collected by Mason got well. The mortality of perforation of the gall-bladder is very high. Twenty-six cases not operated on died; of four operated on, three recovered and one died. For further statistics the reader is referred to Keen's essay.

The following cases illustrating cholecystitis collected by Mason are of interest:

"Case 1. Leudet.[1] Female, aged thirty-six years. Fourth week, pyriform tumor in right hypochondrium, disappearing in ten days; reappearing at intervals during seven weeks. No jaundice. Recovery.

"Case 1. Griesinger.[2] Female, aged twenty years. Sixth week, peritonitis, slight icterus. Painful tumor to right of umbilicus. Swelling of liver; collapse; convalescence. In eighth week sudden return of tumor, with chills, icterus, vomiting; later, two more relapses. Recovery fifth month.

"Case 3. Laveran.[3] Man, aged twenty-three years. Painful symptoms and tumor in region of gall-bladder in sixth week. Recovery.

"Case 4. Martin-Solon.[4] Patient died of peritonitis, and twenty-five ulcers of gall-bladder were found. Previous illness not clearly typhoid fever. Enterite (?).

"Case 5. Husson.[5] Child, aged eight years. Died at end of third week. Perforated gall-bladder. Cystic duct obliterated and converted into fibrous cord.

"Case 6. Dumoulin.[6] Man, aged nineteen years. Third week, constant nausea and vomiting. Enormous tumor in right hypochondrium, extending to left of umbilicus and into right iliac fossa. Resistance like tense hydrocele. Liver raised. Upper limit line of right nipple. Diagnosis: distended gall-bladder. Repeated

[1] Hagenmüller, ibid. [2] Ibid. [3] Ibid.

[4] Bull. Fac. de Méd. de Paris, 1820–'21, vii. pp. 370–375.

[5] Bull. de la Soc. Anat., 1893, p. 104.

[6] Gaz. Méd. de Paris, 1884, 3d series, tome iii. p. 551.

chills, suggestive of hepatic abscess. Coma; death, sixteenth day. Autopsy: typical intestinal lesions of typhoid; tumor, size of head, containing two litres of greenish bile; no gallstones; adhesions with liver, transverse colon, etc.

"Case 7. Archambault.[1] Infant, thirtieth day, signs of intestinal perforation. Death twelve days later. Perforation of gall-bladder; localized peritonitis.

"Case 8. Barthez and Rilliet.[2] Girl, aged twelve years. Sixteenth day, tumor in right hypochondrium, which gradually disappeared. Death, fifty-second day. Autopsy: perforated gall-bladder; circumscribed pus cavity between liver, stomach, gall-bladder, and colon.

"Case 9. Ranvier.[3] Man, aged twenty-eight years. Died during convalescence in the fifth week. Autopsy: limited peritonitis; perforated gall-bladder; right side of abdomen filled with yellowish, opaque liquid. Walls of gall-bladder two or three millimetres thick and infiltrated with pus. A small calculus. Peyer's patches in stage of cicatrization. Author says he cannot explain this point of suppurative election.

"Case 10. L. Colin.[4] Soldier; end of third week of rather mild attack; jaundice, gastro-abdominal pain. Death eleven days later. Autopsy: peritonitis limited by transverse colon, liver, and abdominal wall. Gall-bladder size of goose-egg; perforated. No gallstones. Ducts pervious. Typical intestinal lesions.

"Case 11. C. E. E. Hoffmann.[5] Female, aged twenty-five years, jaundice sixth week; eighth week, sinuses discharging through abdominal wall. Death twelfth week. Autopsy: destruction of gall-bladder. Abscess beneath liver containing twelve gallstones. Lesions of typhoid.

[1] Bull. de la Soc. Anat., 1852, p. 90.

[2] Maladies des Enfants, 1853, 2d edition, vol. ii. pp. 5, 701.

[3] Bull. de la Soc. Anat. de Paris, 1863, 2d series, tome viii. p. 432.

[4] Études Clin. de Méd. Militaire, Paris, 1864, p. 197.

[5] Zerstorung der Gallenblase bei Typhus. Virchow's Archiv, 1868, xlii. 219–222.

"Case 12. O. W. Foot.[1] Female, aged thirty-two years. Died in eighth week. Small abscess between coats of gall-bladder communicating by a narrow orifice with interior. Extensive adhesions of abdominal wall. One cholesterin calculus, twenty-three grains.

"Case 13. Burger.[2] Man, aged forty-one years. Twelfth day, pain, and tumor size of apple in region of gall-bladder; gradual increase in size; chills; no jaundice. Death from peritonitis in fifth week. Perforation of gall-bladder. Adhesions forming cavity filled with pus. No gallstones; no abscess of liver.

"Case 14. P. L. Legendre.[3] Female, aged thirty years. In second week peritonitis at right upper abdomen. Death twelve days later. Autopsy: perforation of gall-bladder. Pus in peritoneal cavity. Three gallstones."

Mason also gives the following references not already quoted in footnotes:

Medical and Surgical Reports of the Boston City Hospital, third series, 1882.

Budd, George: *On Diseases of the Liver*, third American edition, Philadelphia, 1857.

Harley, John: Article on "Typhoid Fever," *Reynolds' System of Medicine*, vol. i.

Pepper, William: *American Journal of the Medical Sciences*, January, 1857.

Guarnieri: "Contributo alla Patogenesi della Infezione Biliari." Ref. Baumgarten's *Jahresbericht*, 1892, S. 234.

Chiari, H.: "Uber Cholecystitis Typhosa." *Prag. med. Woch.*, 1893, No. 22.

Chiari, H.: "Uber das Vorkommen von Typhus Bacillen in der Gallenblase bei Typhus Abdominalis," Eleventh International Medical Congress in Rome. *Zeitschrift für Heilkunde*, 1894, Band xv. S. 199.

[1] Enteric Fever. Abscess in Walls of Gall-bladder. Irish Hosp. Gaz., Dublin, 1874, ii.

[2] Typhus Abdom. mit Perforat. der Gallenblase in die Bursa Omentalis. Deutsches Archiv. für klin. Med., Leipzig, 1873–'74, xii. S. 623–630.

[3] Bull. de la Soc. Anat. de Paris, 1881, 4th series, tome vi. p. 193.

Létienne: "Recherche Bacteriologique sur la Bile Humaine." *Archives de Med.* Experiment, 1891.

Naunyn: *Cholelithiasis*, Leipzig, 1892.

Pisenti: *Archiv für Exper. path. Med. et Pharm.*, 1886. Ref. Brockbank on *Gallstones*, Philadelphia, 1896.

Sherrington: "Experiments on the Escape of Bacteria with the Secretions." *Journal of Pathology and Bacteriology*, 1893.

Blachstein, A. G.: "Intravenous Inoculation of Rabbits with Bacillus Coli Communis and Bacillus Typhi Abdominalis." *Bulletin Johns Hopkins Hospital*, July, 1891, vol. ii., No. 14.

Flexner: "Certain Forms of Infection in Typhoid Fever." *Johns Hopkins Hospital Reports*, vol. v.

Robson, Mayo: "Diseases of the Gall-bladder and Bile-ducts." *British Medical Journal*, March 13, 1897.

Brockbank: Op. cit., p. 130.

Robson, Mayo: Loc. cit.

Monier-Williams and Sheild: *Lancet*, March 2 ,1895.

Malvoz: *Recherche Bacteriologique sur la Fièvre Typhoide.* Paris et Leipzig. Dupré: Op. cit. Dufort: Loc. cit. Gumprecht: *Deutsche med. Woch.*, 1895, No. 14, et seq.

Von Hoffmann: *Untersuchungen über die Pathologisch-anatomischen Veranderungen der Organe beim Abdominal-typhus.* Leipzig, 1869.

Sometimes in typhoid fever the mesenteric and retroperitoneal glands undergo suppuration and break down, causing sepsis. In other instances a subdiaphragamtic abscess forms because of cholecystitis, of suppuration of these glands, or from perforation of the bowel. A case of this character is recorded by Klein[1] of left-sided subphrenic abscess due to typhoid fever, in which the pus contained the specific bacillus. Three litres of pus were allowed to escape by incision. The patient recovered. Keen tells us that this is the only case he could find in literature.

[1] Über die Pyogene Wirking des Eberthschen Bacillus bei Typhuskomplicationen. Inaug. Dissert., Bonn, 1898.

Tungel[1] reports a very interesting case in which a suppurating mesenteric gland near the cæcum caused perforation of the superior mesenteric artery and death from hemorrhage.

Lehman[2] records a case of suppurating mesenteric gland, the pus of which contained the bacillus of Eberth, and Fränkel[3] reports a case of abscess in the abdomen due to this cause four and a half months after the fever. The specific bacillus was found in this pus also.

Other cases have been reported by Michie,[4] Thomson,[5] and Low.[6]

Jaundice complicating typhoid fever is exceedingly rare. Liebermeister met with it twenty times in 1420 cases, Griesinger ten times in 600 cases, Osler not once in one series of 500 cases. Murchison only saw three cases, all of which were fatal. It is caused by catarrh of the ducts, toxæmia, abscess and gallstones with or without cholangitis. Osler,[7] however, records two cases, in one of which the jaundice developed at the onset of a relapse, in the other at the end of the second week. The first case recovered, the second died of toxæmia. Another case of Jaccoud's, studied by Sabourin,[8] was that of a man of twenty-nine years, in the third week of the disease, who had intense icterus, great asthenia and delirium. Death ensued, and at the autopsy the lesions of typhoid fever were found associated with a condition of the liver resembling acute yellow atrophy of this organ.

In the tropics jaundice seems to be a more frequent complication of typhoid fever than in the temperate zone, for Jamieson[9] records nine cases, of which four died.

Sometimes hypertrophic enlargement of the spleen occurs after

[1] Klin. Mittheil aus der Kaiserlich. Hamburg Allegemeine Krankenhaus, 1864.
[2] Centralblatt für klin. Med., August, 1891, 649.
[3] Verhandl. Kongress für inner Med., 1887, 179.
[4] British Medical Journal, 1888, i. 1388.
[5] Glasgow Medical Journal, 1882, xvii. 244.
[6] British Medical Journal, 1881, ii. 122.
[7] Loc. cit.
[8] Revue de Méd., 1882, vol. ii. p 600.
[9] Imperial Maritime Customs Med. Reports, 1891, 37th issue.

typhoid fever. I have seen two cases; one is under my care at present, the other was some years ago, and is shown in Fig. 20.

A number of cases of rupture of the spleen due to the development of an abscess and later exposure and traumatism have been recorded during convalescence in typhoid fever. A case of rupture of the spleen, not due to these causes is, however, reported

FIG. 20.

Splenic enlargement after typhoid fever.

by Santi Flavio,[1] in a man of twenty years, after having been under observation for ten days, suffering from typhoid fever, developed pleural pneumonia with pleural effusion, which required tapping. Two months later the patient suffered from severe pain in the left hypochondrium, the action of the heart became rapid and feeble, and œdema of the left leg was present. After a brief

[1] Gazetta degli Ospitali, 1891, No. 43.

period of improvement the patient was suddenly seized with peritonitis and died, and the autopsy showed that in addition to the peritonitis there had been rupture of the spleen, and that the pus which it contained had been diffused throughout the entire peritoneal cavity. A recent infarction was found in the neighborhood of the rupture, and the intestines showed evidences of an old typhoid fever.

As an interesting illustration of what a patient may recover from during typhoid fever in the way of an accident extrinsic to his disease, a case is recorded by Heath,[1] of a man of twenty-three years, who at the end of the fourth week of his fever swallowed the clinical thermometer which the nurse had placed in his mouth.

A mustard emetic failed to bring away the thermometer, nor did a castor-oil purge cause its discharge from the bowel, but twelve days after it had been swallowed it was passed unbroken and registered a temperature of 104.7°.

Nervous Symptoms in the Far-advanced Stage of the Disease or Following Typhoid Fever. Paralysis complicating typhoid fever or its convalescence may occur in a number of forms, just as paralysis may occur from lesions due to other causes.

It may occur as a local paralysis or monoplegia, as a general paralysis, as a paraplegia, or as a hemiplegia, and it may be due in the first three instances to peripheral neuritis, in the second instance to a myelitis or neuritis, and in the case of hemiplegia to cerebral lesions, such as thrombosis, embolism, hemorrhage, and meningo-encephalitis. Sometimes the monoplegia or partial paraplegia may be due to a poliomyelitis.

By far the most common of these affections is the loss of power due to neuritis, a condition which is not commonly met with as a complication of typhoid fever, yet not so rare as the other changes just named. The most exhaustive and interesting monograph concerning this complication of the disease is that given us by Ross and Bury,[2] in their essay on "Peripheral Neuritis," first published

[1] American Lancet, December, 1888.

[2] A Treatise on Peripheral Neuritis. Griffin & Co., 1893.

in the *Medical Chronicle* and afterward in a separate volume. So complete and thorough is their study of the literature of the subject and of the clinical aspect of the condition that much of the following information is to be credited to them.

Gubler,[1] among several cases of local palsy after typhoid fever, records the case of a boy of sixteen years, who developed, a few days after his fever ceased, a nasal voice, which was found to depend upon paralysis of the palate. Shortly after this there was paralysis of accommodation. This latter point is of interest in view of the fact that Gowers states that this condition never arises from typhoid fever. Gubler also states the case of a boy who, after an attack of forty-seven days, suffered from paresis in his legs and became unable to raise himself in bed. His lower limbs were feeble, tremulous and their muscular irritability greatly increased. There was also loss of power in the hands with some spastic contraction of the fingers, and the speech was staccato.

Surmay[2] records two cases of local paralysis due to this cause. In one the loss of power was in the extensor muscles of the hand and fingers and in the extensors of the toes, and in the other case, weakness of the right leg was followed by complete loss of power in the left. So, too, Kraft-Ebing[3] speaks of weakness of the adductors of the thigh and hyperæsthesia of the skin supplied by the saphenous nerve. Bailly[4] has recorded paraplegia, anæsthesia, and contractions in these cases, and in two instances paralysis of the palate, and Nothnagel[5] records four patients in whom the ulnar nerves were paralyzed and the ulnar side of the hand was anæsthetic. In all these cases there was the reaction of degeneration, and they also suffered from radiating pains in the upper and lower extremities. In four other cases there was partial paralysis of the lower limbs with partial anæsthesia, pain, and tingling sensations, and in one of these patients the trouble in the lower

[1] Gubler. Arch. Général de Méd., 1860.

[2] Surmay. Arch. Général de Méd., 1865, tome i. p. 678.

[3] Kraft-Ebing. Beobachtungen und Erfahrungen über Typhus Abdominalis, 1871.

[4] Bailly. Thèse de Paris, 1872.

[5] Nothnagel. Deutsches Arch. für klin. Med., Bd. ix. p. 429.

extremities was followed by weakness in the upper limbs. In still another the patient at the beginning of convalescence first had a feeling of numbness and creeping in the left leg, and after this, paralysis of that limb gradually developed. Later on the extensors of the right hand became paralyzed, and four days later some of the muscles of the left hand.

Similar cases have been reported by Leyden[1] and Benedict, and in one recorded by Eisenlohr,[2] a man of thirty years, eleven days after his temperature became normal, suffered from numbness and loss of power in the left leg and feet, with violent pain in these parts and in both knees, followed the next day by effusion into the right knee and a rise of temperature to 104°. There was loss of power in the left peroneal nerve, and fourteen days later the left knee became swollen. On the sixteenth day the right elbow became swollen and painful and the swelling of the left knee subsided. The muscles supplied by the left peroneal nerve showed diminished reaction, and the left foot was œdematous and in the position of equino varus. On the twenty-fourth day the flexors of the feet and the extensors of the toe were completely paralyzed, and gave the reaction of degeneration.

This case of Eisenlohr's is of interest, first because the swelling passing from joint to joint might have aroused a suspicion that the cause was rheumatic, and because certain writers in quoting the case considered it as an instance of paralysis coming on during relapse. As Ross and Bury point out, it is possible that the rheumatic poison was the cause of both the joint changes and the evidences of neuritis.

Additional cases of peripheral neuritis have also been reported by Bernhardt,[3] Vulpian, and others. Thus a case of deltoid paralysis has been recorded by Vulpian,[4] which was in all probability due to a peripheral neuritis. A young man of eighteen years, after an attack of typhoid fever, suffered from pain in the arm

[1] Leyden. Klinik de Ruckenmarkskrankheiten, 1875, Bd. ii. Abth. 1, p. 247.

[2] Eisenlohr. Arch. für Psychiatrie und Nervenkrankheiten, 1876, Bd. vi. p. 543.

[3] Bernhardt. Deutsch. Arch. für klin. Med., 1878, p. 363.

[4] D'Accident Survenus Pendant la Convalescence de la Fièvre Typhoide. Revue de Médicine, 1883, p. 617.

and developed loss of power in the right shoulder, with atrophy of the deltoid muscle. In none of these cases, however, were any studies made, over and above the clinical tests which are ordinarily employed, to prove positively that a true neuritis was present, and it was not until Pitres and Vaillard[1] published their paper, in 1885, that the first careful microscopical observations upon typhoid peripheral neuritis were presented. After detailing the cases of two patients who suffered from typhoid neuritis they give the results of the histological examination of nerves removed from the bodies of four patients who died during the active period of typhoid infection, but in whom no signs of peripheral neuritis had been noted during life. Curiously enough, in three out of these four cases changes indicating parenchymatous neuritis were found to be present, and, still more curiously, one of these patients died as early as the sixteenth day of the disease, while two others died on the thirty-sixth and twenty-fourth days respectively.

Other instances of post-mortem examinations revealing peripheral neuritis in typhoid fever are those reported by Oppenheim and Siemerling. In one of these instances the patient died in the middle and the other at the end of the second week of the fever, and in both cases parenchymatous degeneration of the peripheral nerves was found, in one of which it affected the great saphenous and peripheral nerves, and in the other a branch of the cutaneous nerve supplying the dorsum of the right foot, and showed complete degeneration of many of its fibres.

Since these papers have been published, others dealing with the clinical aspect of the case have been placed upon record by Alexander,[2] Handford,[3] Archer,[4] Humphreys,[5] Klumpke-Déjèrine,[6]

[1] Pitres and Vaillard. Compte Rendu. Soc. de Biol., Paris, 1885, S. 8, ii. 661, and Rev. de Méd., Paris, 1885, v. 985.

[2] Alexander. Deutsche med. Wochenschrift, 1886, vol. xii. 529.

[3] Handford, H. Peripheral Neuritis in Enteric Fever. Brain, vol. xi. 237.

[4] Archer. British Medical Journal, 1887, vol. i. p. 727.

[5] Humphreys (F. R.). A Case of Peripheral Neuritis following Typhoid Fever. Abstr. Tr. Hunterian Society, London, 1889–90, 41.

[6] Déjèrine-Klumpke. Des Polynévrites en Général et des Paralysies et Atrophies Saturnines en Particulier. Paris, 1889, p. 222.

and notably the two cases reported by Bury in the essay which I have named. One of these was in a girl of eighteen years, who was seen eight months after an attack of typhoid fever of varied duration and severity. During the fever she was suddenly affected by a condition in which she was unable to straighten out her upper and lower limbs, and this rigidity persisted until she was admitted to the Manchester Royal Infirmary, eight months afterward, when it was found there was great wasting of all the muscles of the limbs, particularly in the muscles on the front of the thigh and outer part of the legs. There was drooping of the great toes and the knee-jerks were variable, sometimes being excessive and sometimes being minus. The plantar reflexes were absent, and there was no ankle-clonus. The upper limbs were somewhat flexed, and could not be extended, and there was atrophy of the thenar and hypothenar eminences; there were also marked disorders in cutaneous sensibility in the distribution of the radial nerve. The contractions could not be overcome even when the patient was put under chloroform, and while the paralysis and rigidity remained for many weeks, the patient ultimately made a complete recovery.

In still another case, long after typhoid fever, a man of forty-two years, suffered from pains in his legs, in which all the muscles below the knees presented a moderate degree of wasting; he had exaggerated knee-jerks.

Dercum has reported to the author two cases of peripheral neuritis after typhoid fever, due to the excessive administration of alcohol during the illness. Thus a girl of fourteen years received one and a half pints a day for some time, and developed typical alcoholic neuritis.

These cases give some idea of the character of the various forms of peripheral neuritis which follow typhoid fever. Other instances might be quoted in which there was doubt as to whether paraplegic symptoms were due to neuritis or to damaged tone of the tracts and cells in the spinal cord. Thus Mitchell[1] has recorded a case

[1] Mitchell (S. W.). Boston Medical and Surgical Journal, 1879, c. 245.

of paraplegia associated with tremor, in which he thought that the paralysis was due to degeneration of the cells in the anterior cornua of the spinal cord, but Ross and Bury consider that the rapid improvement of this patient indicated that she was suffering rather from a peripheral than a spinal disease. So, too, George Ross[1] has recorded a case in which there was paralysis with spastic contraction of the lower extremities, with loss of electrical reaction, but no diminution in the abilities of the sphincters, and in which complete recovery took place.

That severe peripheral neuritis may result in trophic changes in the organs supplied by the nerves which are involved is shown by a case reported by Wedenski,[2] of a boy of seventeen years, in whom, two years after typhoid, symmetrical gangrene developed as a result of degeneration of the peripheral nerves. No lesions were found in the muscles nor in the cerebro-spinal nervous system.

Closely associated with the question of true paraplegia following enteric fever is that partial paraplegia or ataxia of the stage of convalescence in which there is a strange inability of the patient to use his lower limbs. This lasts in nearly all severe cases for some days after the patient leaves his bed, and is often persistent for some weeks, causing a peculiar waddle or stiff-legged gait quite pathognomonic of this state.

In connection with the question as to whether these various forms of paralysis are spinal or peripheral, the following quotation from Ross and Bury is of importance:

"While it is probable that a few cases of muscular atrophy which follow typhoid fever depend upon an anterior poliomyelitis, and that a condition similar to that of infantile paralysis is produced, the presence of sensory disturbances in the vast majority of cases shows that the lesion, if in the cord at all, is not limited to the anterior horns, or involves both the anterior and posterior roots, or the mixed peripheral nerves. The absence of spinal

[1] Ross (G.). International Journal of the Medical Sciences, 1889, p. 25.

[2] Wiener Medizinischer Presse.

tenderness, of girdle pains, and of disturbances of the sphincters speaks much against an infection of the spinal cord or its roots, while the initial sensory disturbance, succeeded by a limited paralysis having a slow progressive march up to a certain degree, which varies according to the severity of the case, the paralysis then slowly receding and ultimately, as a rule, completely disappearing, are points strongly in favor of an affection of the peripheral nerves."

An interesting case of peripheral neuritis after typhoid fever has been recorded by Putnam, of Boston. In this the patient suffered from trophic changes in that small abrasions did not heal. There was marked analgesia, and when seen two years after the attack of the fever, this disturbance of sensation extended to the left arm and shoulder, the left side of the neck and trunk as far as the eighth rib. Marked improvement followed treatment.

There are three other classes of symptoms showing peripheral-nerve disturbances: First, cases in which excessive muscular contractions are developed in place of paralysis, but associated with pain and hyperæsthesia. Eleven of these cases have been reported by Aran in *L'Union Médicale*, July, 18, 1855. The contractions occurred toward the end of the attack of typhoid fever, and never were begun with the commencement of the disease. They were preceded by formication, prickings, and numbness in the extremities, and pain in the joints, and the immediate seizure was associated with an intense feeling of anxiety and distress, the contractions affecting both upper and lower limbs, so that many muscles exhibited almost incessant fibrillary contractions. By gradual manipulation, artificial extension could be obtained, and this gave the patient relief for a short time. In four cases the muscles of the trunk were affected and opisthotonus was produced, the patient being held immovable by the muscular contraction, which also caused great pain. These attacks lasted from a quarter of an hour to three hours and recurred from two to ten times a day, and after the cessation of the attacks the fever ran its ordinary course without any other symptoms save an occasional numbness of the affected parts. Although three of the patients died, Aran thinks

their deaths were due to the severity of the fever and not to the tetanic complication. These cases so closely resemble tetanus that similar ones could be readily taken for tetanus if the symptoms occurred early in the course of typhoid fever.

Gubler[1] has recorded a case of contraction of the hands, and Dewèrve refers to this condition as possible of occurrence in the *Nouveau Dictionnaire de Médicine et de Chirurgie.* So, too, Nothnagel[2] refers to a case of tonic contractions of the interosseous muscles lasting from one-quarter to one-half an hour. Similar contractions ensued when the patient supported himself on his toes.

A second class of nervous disturbances is closely associated with the general signs of peripheral neuritis, and is thought by some to have become more frequent since the general introduction of the cold bath in the treatment of typhoid fever. These signs have been particularly described by Handford, and consist of great hyperæsthesia of the toes and heels of patients in the latter part of the disease or, more particularly, during convalescence.

Finally, a few cases have been recorded in which the rapidly ascending paralysis, usually terminating fatally, has occurred during the course of, or immediately after, an attack of typhoid fever.

Cases of myelitis or anterior poliomyelitis, as a result of typhoid fever are so rare as to be almost unknown, although Gowers, as already quoted, has stated that poliomyelitis is more frequently secondary to typhoid fever than to any other acute infectious disease.

Two cases of ascending myelitis are recorded by Raymond in *La Science de Médicine* for 1885, but in each of these there is good reason to believe that the lesions were really those of neuritis and not really those of myelitis. A case has, however, been reported by Shore in the *St. Bartholomew's Hospital Reports,* vol. xxiii., in which there was acute myelitis of the anterior cornua and involvement of three of the eight cervical nerves.

[1] Archives Générale de Méd. xv. 5th series.

[2] Deutsche Arch. für klin. Med., 1872, 9.

Hemiplegia arising from typhoid fever is not as rare as myelitis, and is far less common than paralysis due to peripheral neuritis. By far the most extensive research into the literature of this subject is that of Dr. Francis Hawkins, who has collected in the *Clinical Society's Transactions* for 1893, vol. xxvi., 17 cases from literature; 3 of these occurred in children under fifteen years of age, and the time of onset in 14 of the cases was the second week; in 1 case the third week; in 6 cases the fourth week, and in 5 cases during convalescence. The right side was paralyzed in 12 of the 16 cases in which the statement as to the side paralyzed was given, and aphasia occurred in twelve instances. Curiously enough, only two of the seventeen cases died, and in both of these a thrombus plugged the middle cerebral artery. In all probability a great majority of the cases of hemiplegia complicating typhoid fever are due to this lesion. Thus, Osler has recorded a case of a young physician who was taken ill with typhoid fever, on the fourteenth day had a temperature of 104°, which, however, fell the following morning to 100.7°, and in the next three or four days the temperature had not reached 102.5° when the rash developed and the spleen became palpable. Twenty-four hours later, when all the symptoms of the case seemed favorable, he was suddenly seized with uneasy feelings in his head, the pupils were dilated, and in a few minutes he suffered from a short, sharp general clonic convulsion, beginning almost simultaneously in both arms; the eyes showed marked conjugate deviation to the left and upward, and the head was also turned to the left. The convulsions were profound at short intervals for an hour, then became less intense, and finally ceased altogether for several hours; they were accompanied by profound unconsciousness, and the severer ones occasioned great embarrassment to the respiration. In the interval the patient was conscious, spoke to those about him, and seemed to understand questions. Later in the evening the convulsions recurred with great severity, and after five hours the patient died in a severe one. These convulsions were general, but were most marked on the right side of the body. A post-mortem examination held by Flexner revealed thrombosis in the ascending

parietal and parieto-temporal branches of the middle cerebral artery. The meninges over these vessels contained small hemorrhages, and the brain-matter, while not softened, showed small extravasations of blood. Small but quite extensive punctiform hemorrhages could be seen to occupy the cortex and adjacent white substance in the immediate neighborhood of the thrombosed vessels.

Out of the well-known 120 cases collected by William Osler of hemiplegia in children there was no instance of hemiplegia following typhoid fever, and in 160 cases collected by Wallenberg, four only occurred after typhoid fever. Osler,[1] however, reports two cases of post-typhoid hemiplegia. One of these occurred in a girl of six years. Almost two months after the beginning of her illness she was seized with violent convulsions, which were confined to the head, right arm and leg; she became unconscious. Later it was noticed that the right side was completely paralyzed, including the face, and that there was total loss of speech and aphasia, lasting for seven weeks. Gradually the patient largely recovered from this paralysis, but complete recovery did not ensue. The second case was that of a clergyman, aged twenty-five years, who was seized with convulsions fourteen days after going to bed with headache, fever, and diarrhœa. In this case also partial recovery took place, but Osler did not, at the time of making his report, consider that complete recovery would be possible. The paralyzed arm, the left, many months after the attack, was affected by wide irregular choreiform movements on attempting any voluntary effort, but his mental condition was excellent.

Another case of this character was reported to the Johns Hopkins Medical Society by Blumer:[2] that of a little girl who one week after convalescence had begun, and who had been eating solid food, was seized with violent convulsions, which were confined almost entirely to the right side. These convulsions lasted for eight hours, and were followed by paralysis of the right side;

[1] Journal of Nervous and Mental Diseases, May, 1896.

[2] Johns Hopkins Hospital Bulletin, April, 1896, p. 72.

five weeks after the onset of these convulsions she began to recover both the power to move the arm and leg, and also that of speech; she suffered from amnesic aphasia; ultimately almost complete recovery took place, so that there was only slight dragging of the foot, and some pure motor aphasia. The arm, however, did not materially improve, and was affected by rigid paralysis, though with no sign of facial paralysis, and the tongue was protruded straight. Blumer believed that the case was due to thrombosis.

In the same journal Thayer records two other cases of this character seen in the Massachusetts General Hospital. On the tenth day of the illness in one case the ward orderly found at 1 A.M. that the patient was unable to move the right arm and leg; the face was flushed, the eyes half closed, the pupils equal, and eyeballs rolled upward. The patient's mental condition was very stupid. Eight days later the patient was distinctly better, unable to speak, but evidently understood what was said to him; he could not protrude his tongue, but later was able to read the paper and to say a few words.

The other case was that of a girl of ten years, admitted to the Massachusetts General Hospital on the fifth day of typhoid fever, who was found on the twenty-third day of her disease to lie principally upon the right side, and failed to answer questions. The next day the patient could not speak, although she apparently understood what was said to her; the tongue was protruded straight; the face was not paralyzed.

In other words, these are two cases illustrating the onset of complete right-side hemiplegia with motor aphasia.

A case of hemiplegia has also been recorded by Newbolt,[1] in which a locomotive fireman of twenty-one years suffered from loss of power in the left arm and leg during the course of a relapse. There was aphasia, and the tongue was protruded to the right; there was drooping of the right eyelid, and some dysphagia. Perfect recovery did not occur. The case was thought to have been due to thrombosis.

[1] London Lancet, August 27, 1893.

Still another case of hemiplegia complicating typhoid fever is recorded by Imradi.[1] The case had been considered one of influenza, and the patient was allowed to go out on the fifteenth day, when he suddenly lost consciousness and remained unconscious for hours; when seen he was suffering from left-sided hemiplegia. The fever ran a typical characteristic course, but recovery occurred.

Imradi asserts that there are only fifteen similar cases to be found in literature.

Vulpian[2] has recorded a case of obstruction of the left Sylvian artery in the course of typhoid fever, causing right hemiplegia and aphasia in a male of seventy years.

Under the title of "A Case of Hemiplegia of Gradual Onset Following a Severe Attack of Enteric Fever, and Terminating in Insanity" (which was probably male hysteria), Stevens[3] has recorded the history of a man of twenty-two years who three months after recovery from this disease found he had difficulty in approximating the fingers of his left hand to one another. He tells us that "the fingers are flexed upon the palm of the hand more or less. They can passively and slightly, by voluntary effort, be extended within narrow limits (see figure in *Glasgow Medical Journal*). The thumb is turned outward and flexed at the interphalangeal joint. Forcible extension of the fingers is accompanied by considerable pain, but the thumb is less painful in this respect. The wrist joint is fixed, evidently largely by muscular spasm, and not by definite anchylosis. Movement of flexing the forearm on the arm is perfectly easily accomplished, but it is accompanied by considerable fine tremor of the whole arm. On attempting to raise the left arm above the head it becomes evident that there is little movement at the shoulder-joint. Most of the movement is accomplished by moving the arm and shoulder *en masse*, and, as a result the range is much more limited than on the other side. There is no definite wasting of any of the arm muscles. The position of the thumb in relation to the other fingers is

[1] Centralblatt für de med. Wissenschaften, October 25, 1891.

[2] Revue de Médicine, 1884, p. 162.

[3] Glasgow Medical Journal, January to July, 1897, vol. xlvii.

further noted. It is turned around in such a way that it rests upon the radial aspect of the first phalanx of the forefinger. As regards the foot, there is noted a spastic condition evidently involving the extensors, so that the toes are all drawn well up upon the dorsum of the foot, the first phalanx in each case being drawn far back upon the metatarsal bone. The extensor tendons stand out like cords. Despite this, movement of the ankle-joint is fairly free, although rather jerky. The power of the muscles of the thigh, as tested by making and resisting movements of flexion and extension of the knee, is fairly good in both lower extremities, and no appreciable difference is made out between the two sides.

"Sensation is tested in both upper and lower extremities, and found to be normal. The reflexes (tendon) in the left upper extremity are abolished; in the right, normal. The superficial abdominal and cremasteric reflexes on the right side are easily elicited; the former can be faintly brought out on the left side, but the latter on the left side cannot be elicited. The knee reflex is distinctly exaggerated on the left side, and the ankle-clonus is very marked, while on the right side the knee reflex is normal, and there is no ankle-clonus."

Later the patient became insane and passed into an asylum, and the asylum physicians made the following report on his case, deciding that the condition was male hysteria. They state:

"The points that guided us in inclining to a diagnosis of the hysterical nature of the case were as follows:

"1. The varying intensity of the symptoms. The flexion of the arm was not constant; at times it admitted of a limited movement and a limited power of passive extension, but at other times the spasm of the flexors was intense, and manipulation was almost consciously resisted. The symptoms in the leg varied even more than in the arm.

"2. The comparative absence of atrophy of muscles, considering the duration of his illness (since the middle of 1895). Measurements taken last month showed that while there was a degree of atrophy the greatest difference was between the right and left thighs, which was only one and one-quarter inch.

"3. Apparently normal response of the muscles to faradic irritability.

"4. The complete disappearance of the symptoms under deep chloroform necrosis.

"There were also the peculiar hysterical posture of the patient and the difference between the symptoms in the two limbs."

Still another case of hemiplegia is reported in the *Johns Hopkins Hospital Bulletin* for July, 1896, by Haynes, as having presented itself at the Brooklyn Eye and Ear Hospital. A man of thirty years suffered in October, 1895, from an attack of typhoid fever lasting twenty-one days. On the fourteenth day his left arm became paralyzed, and when able to sit up it was found that both upper and lower extremities felt numb, although there was no loss of sensation. This condition persisted for a couple of months, when improvement began, first in the leg; almost complete recovery ensued so that only slight loss of motion and inability existed. There was no evidence of facial paralysis or convulsions in this case.

As an indication of the possible effects of embolism of the cranial vessels, the case recorded by Mensel may be cited, in which necrosis of the skull followed the formation of a clot in the middle meningeal artery.

Aphasia or other disturbances of speech after enteric fever have also been recorded by a number of observers without simultaneous hemiplegia. Thus Hutinel[1] tells us that aphasia always occurs in children, and more frequently in boys than in girls. In some of these instances the condition arises from embolism, but in other cases recovery has ensued so rapidly that no severe organic cause of this character could have been present, and this has been proved by the failure to find embolism at autopsy. Leyden has expressed the view that such cases may be due to a mild degree of encephalitis with readily absorbed exudation.

Mental disturbance following typhoid fever is by no means rare, and varies in degree from slight mental enfeeblement and inability

[1] Étude sur la Convalescence et les Rechute de la Fièvre Typhoïde, Paris, 1883.

to do mental work to marked insanity. When the patient is violent they are said by some persons to have "asthenic mania." It is not mania, but the insanity of profound mental and physical depression. These variations from the normal are usually followed by recovery, as is pointed out in the interesting chapter on the mental disorders of the late stage of typhoid fever, which has been contributed to this essay by the author's friend and colleague, Dr. F. X. Dercum, Clinical Professor of Diseases of the Nervous System in the Jefferson Medical College.

Rathery[1] and Hutinel have recorded cases of post-typhoid tremor. In one of Rathery's cases it persisted fifteen months after the fever ceased. Similar cases have been recorded by Freund.[2]

Fry,[3] of St. Louis, records a case of so-called paralysis agitans following immediately after typhoid fever. The trouble began with the ending of the fever in a tremor, which gradually increased in violence, and chiefly involved the right arm and later the left. Still later the legs were involved. No definite reason for believing the case to be Parkinson's disease and not one of ordinary tremor is vouchsafed.

Gubler[4] has recorded amaurosis and strabismus after typhoid fever, and the latter symptom has also been seen by Nothnagel.[5] Paralysis of the soft palate has also been recorded by Gubler, and of the vocal cords by Turck and Nothnagel. All these symptoms are but evidences of the peripheral neurites already discussed.

Bouley and Mendel[6] state that paralysis of the vocal cords following typhoid fever is, in their opinion, an exceedingly rare condition. They claim they have only found ten other cases in literature which are carefully described and three others briefly mentioned. In some of these cases there was complete paralysis of the recurrent laryngeal nerve with profound paralysis of the adductors. Bernoud[7] has also reported cases.

[1] Des Accidents de la Convalescence, Paris, 1875.
[2] Inaugural Dissertation, Breslau, 1885.
[3] Journal of Nervous and Mental Diseases, 1897, p. 465. [4] Loc. cit.
[5] Loc. cit. [6] Archives Générale de Médecine, December, 1894.
[7] Lyon Médicale, March 28, 1897, p. 453.

Paralysis of the laryngeal muscles is probably more common than is generally thought, arising, as a rule, from neuritis. Thus Przedlorski found in 100 consecutive cases no less than 25 cases with paralysis.

Very recently, at a meeting of the Laryngological Section of the College of Physicians of Philadelphia, Dr. MacCoy reported three cases of this rather rare condition of laryngeal paralysis complicating typhoid fever. As he well said in his preliminary remarks :

" We can most simply classify these paralyses under the various functions performed by the larynx. Keeping clearly in mind that the chief function of sets of laryngeal muscles is to open and close the glottis, we can simplify the clinical facts by grouping them under the two heads of paralysis of adduction and of abduction. Paralysis of adduction in its various forms is of very great interest, and enters largely into our most interesting laryngological experiences ; but it concerns phonation only—a most wonderful function, but not necessary to life. Abduction, on the other hand, concerns the very existence of life—respiration. A moment's faltering in the function of the openers of the larynx, and we cease to exist. Being, then, of so vital importance, we must promptly recognize, during the course of a prolonged and wasting acute disease like typhoid fever, the imminent risk to life when the abductor muscles are paralyzed."

Dr. MacCoy has been good enough to send me the following reports of his cases for mention in these pages :

The first case he saw was one of posterior crico-arytenoid paralysis. It was double or bilateral, and occurred in a case of typhoid fever at a suburban hospital. The subject was a young man who had had a severe, prolonged and complicated attack. The patient had been ill for over two months, was greatly emaciated, and profoundly debilitated. One night he was suddenly seized with a suffocative attack simulating croup. Getting no relief whatever from remedies applied, Dr. MacCoy was asked to see the case. The patient was greatly distressed in his respiration and cyanosed. Inspiration was performed laboriously, each inspiration being

accompanied by stridor, and the patient appeared almost moribund. Laryngoscopic examination showed a complete double paralysis of the openers, the vocal bands remaining fixed in the median line. Accompanying paralysis of the arytenoid muscles with loss of tension enabled the patient to get a little air through a small triangular slit at the most posterior portion of the glottis. As promptly as possible an adult intubation tube was inserted into the larynx. This was accomplished without much distress or trepidation to the patient. The effect of the intubation was magical; complete relief to breathing instantly followed, and in a few minutes the patient was in a quiet sleep.

The second subject presented himself for consultation. He was a young man of twenty-three years. He wore a tracheotomy tube. The history showed that he had had a severe attack of typhoid fever in the South a few months previously. During convalescence he was seized with a grave suffocative attack, and was in such a serious condition as to require tracheotomy, which relieved him completely. Examination of the larynx showed a complete fixation of the vocal bands in the median line. This patient could not do without the tube, and he requires it to the present time. He has a most clever device of a valve and rubber tubing and rubber bulb connected with the canula, by which air is made to close the valve against the mouth of the canula, and so he is enabled to carry on conversation with ease and fluency. In this case intubation was attempted but failed of introduction. The subject enjoys good health and is active in business pursuits.

The third case was a soldier in one of the city hospitals, who was suffering from great dyspnœa. Laryngoscopic examination showed complete apposition of the vocal bands in the median line with enough relaxation of tension and arytenoidal paralysis to allow a little air to enter. Intubation was strongly urged, but the visiting physician was relucant, and the subject died of exhaustion in a short time. In MacCoy's judgment, prompt intubation in this case would have saved the man's life.

Finally, cases of chorea have been recorded by Rilliet and

Barthez, but these may have been cases of tremor rather than chorea.

Sometimes in the convalescence a curious state is developed in which the muscles of the lower extremities become painful, somewhat brawny, and even slight redness may appear in the skin covering them. Usually this is unilateral, but it may be bilateral. Most commonly it affects the calf of the leg, and pain is developed on pressure or on movement, active or passive. Osler believes this to be a myositis. Whatever it may be, the author can indorse the statement that the condition is painful, from his own experience, although the condition was not well developed.

Many years ago V. P. Gibney, of New York, described under the name of "typhoid spine," a condition in which there develops, often some days after the patient is up and about, and often only after some very slight jar or trauma, great tenderness of the spine, and pain in the back and in the legs when they are moved. This condition is not dependent upon a spondylitis, neuritis, or Pott's disease, and is probably a neurosis closely allied to the neuroses seen in severe cases of trauma.

Sometimes neurotic patients, particularly women, suffer from hysterical attacks of causeless weeping while convalescence progresses, and in a case under the writer's care, during convalescence, a strong and hearty man, a member of the city fire department, cried like a child whenever one of his fellows came to visit him.

Severe hysteria sometimes complicates convalescence in typhoid fever. Thus Simpson[1] records the case of a woman who was suddenly seized with unconsciousness and rigidity during convalescence; she was confined to bed for nine years, but had regular attacks on each succeeding Sunday, the day on which the first attack occurred. Constant vomiting was also present.

A condition of very great rarity after enteric fever is tetany. Janeway has reported cases coming on during the height of typhoid fever, the tenth and twenty-fourth days.

Pseudo-hypertrophic muscular changes have been recorded as

[1] Edinburgh Medical Journal, January, 1896.

occurring after typhoid fever by Lasage.[1] The patient, a man of twenty-seven years, was seized on the nineteenth day of the attack with acute pain in the left thigh and with other symptoms, which caused a diagnosis to be made of phlegmasia. Swelling of the limb did not, however, disappear, and several months later it was found to be greatly increased, the hypertrophy involving the muscular masses, which were larger and firmer than in the right leg, although the electrical reactions were not impaired, nor were the reflexes. Exercising the muscles on this side produced cramp-like contractions. At the time the case was reported the condition had persisted for two years.

The following references for which I am indebted to Ross and Bury's monograph, may be of interest in this connection:

Meyer. *Die Elektricität auf Praktische Medicin,* Berlin, 1861, p. 311.

Leudet. "Remarques sur les Paralysies Essentielles Consécutives à la Fièvre Typhoïde," *Gaz. Méd. de Paris,* 1861.

Imbert-Gourbeyre. "Recherches Historiques sur les Paralysies Consécutives aux Maladies Aigues," *Gaz. Méd. de Paris,* 1861.

Handfield-Jones. "Abstract of a Clinical Lecture on a Case of Paralytic Contracture after Fever," *Medical Times and Gazette,* 1867, p. 390.

Murchison. *A Treatise on the Continued Fevers of Great Britain.* Second edition, 1873, p. 225.

Teale and Morven, quoted by Nothnagel. *Deutsche Archiv f. klin. med.,* 1872.

Rehn. "Ein Fall von Lähmung der Glottiserweiterer nach Typhus Abdominalis," *Deutsches Arch. f. klin. Med.,* Bd. xviii. p. 136.

Landouzy. *Des Paralysies dans les Maladies Aigues.* Paris, 1880.

Baumler (C.). "Ueber Lähmung des Musculus Serratus Anticus major nach Beobachtungen an Cinem Fall von Multiplen Atropischen Lämungen im Gefolge von Typhus Abdominalis," *Deutsches Archiv. f. klin. Med.,* 1880, vol. xxv. p. 305–324.

[1] Revue de Médecin, November 10, 1889.

Stintzing (R.). "Typhus Abdominalis mit Nachfolgender Atropischer Lähmung," *Aertztl. Int. Bl.*, Munchen, 1883, vol. xxx. p. 4.

Bartholow (R.). "Enteric Paraplegia," *Medical News*, Philadelphia, 1883, vol. xliii. p. 609.

Rondot (E.). "Contribution à l'Étude des Paralysies qui Surviennent dans la Fièvre Typhoïde; Paraplégie et Amyotrophie Myelopathiques d'Origine Typhoïdique," *Gaz. Hebd. de Sci. Méd. de Bordeaux*, 1885, vol. vi. p. 446.

Péliotis. *De la Névrite Périphérique du Cubital Consécutif à la Fièvre Typhoïde.* Paris, 1885, Thèse.

Raymond. "Deux Cas de Myélite Ascendante Observés pendant la Convalescence de la Dothiénentérie," *Revue de Médicine*, 1885, p. 648.

Courtade (D.). "Des Paraplégies Survenant dans le Cours ou pendant la Convalescence de la Fièvre Typhoïde," *L'Encéphale*, Paris, 1886, vol. vi. p. 431.

Würtz. "Note sur un Cas de Névrite Tibial Antérieur Survenue dans le Cours d'une Fièvre Typhoïde," *L'Encéphale*, 1886.

Buzzard (T.). *Paralysis from Puerperal Neuritis*, 1886, p. 102.

Bassi (U.). "Névrite Multipla Consecutiva a Febbre Tifoide," *Rév. Veneta di Sc. Med.*, Venezia, 1887, vol. vi. p. 585.

Oppenheim and Siemerling. "Beiträge zur Pathologie der Tabes Dorsalis und der Peripherischen Nervenerkrankung," *Archiv für Psychiatrie*, 1887, p. 509.

Puybaret (J. A. C.). *Contribution à l'Étude des Paralysies dans la Fièvre Typhoïde*, Bordeaux, 1887, Thesis.

Stadelmann. "Ueber einen Eigenthümlichen Mikroskopischen Befund in den Plexus Brachialis bei einer Neuritis in Folge von Typhus Abdominalis," *Neurol. Centralb.*, 1887, p. 285.

Gowers. *A Manual of Diseases of the Nervous System*, vol. ii. p. 824.

Stoney (W.). "Paralysis of Extensor Muscles of Thigh Following Enteric Fever," *Medical Press and Circular*, 1889, N. S., vol. xlvii. p. 562.

Kebler (J.). "Post-typhoid Paralyses," *Cincinnati Lancet-Clinic*, 1889, N. S., vol. xxiii. p. 35.

Longstreth (M.). "Neuritis after Typhoid; Rheumatic Neuritis," *Physician and Surgeon*, Ann Arbor, Mich., 1887, vol. ix. p. 201.

Comte. "Un Cas de Paralysie Généralisée à la Suite de la Fièvre Typhoïde," *Poiteau Méd.*, Poitiers, 1887, Tome ii. p. 113.

Schmidt (F.). "Ueber Neuritsche Lähmungen nach Abdominaltyphus," Nürnberg, 1891.

Pal. "Uber Multiple Neuritis," Wien, 1891, p. 37.

The Skin in the Stage of Convalescence. Aside from boils, carbuncles, and gangrene, which may appear at this time, and which have been discussed under the heading of the well-developed stage of the malady, we find as the most common complication at this time erysipelas.[1]

According to Liebermeister, this complication occurs generally during convalescence and seldom at the height of the disease, and he believes it may be a dangerous factor. In 1420 cases of typhoid fever in Basel, erysipelas appeared ten times, and all of the ten recovered. These were all cases of facial erysipelas. Two others developed the disease about bed-sores. In other words, erysipelas occurred in a little less than 1 per cent. of these cases. Griesinger[2] states that it occurs in about 2 per cent. Taupin (1839) speaks of two cases of erysipelas of the face occurring in children suffering from typhoid fever.

The following cases occurred within a period of six weeks of each other in the wards of St. Agnes's Hospital under my care. The first case was separated from the second by an interval of five weeks, and the second from the third by less than a week. They were all in the same ward, but occupied beds at least twenty feet apart. The first case is as follows:

Maggie T., aged twenty-two years, was admitted December 16, 1890, with a history of chronic suppuration of the middle ear. She was treated at the dispensary, and rapidly improved, being discharged on December 23d. On January 8, 1891, she was re-

[1] See article by Hare and Patek in the Medical News, January, 1891.

[2] Infectionskrankheiten.

admitted with well-defined symptoms of a mild attack of typhoid fever, which ran a short course, the patient being discharged on January 30th. On February 2d she entered the house, complaining of pain in the abdominal region and in the knees and elbows; the pains were not very severe, but the joints were somewhat swollen; the tongue was brown and dry, and all the symptoms, such as the stools, the rose-colored spots, the characteristic temperature and appearance of the patient, pointed to a second attack of typhoid fever, although at first the case was treated as one of rheumatism. The temperature did not exceed 103°, and the patient went through a moderately severe attack of typhoid fever without complication, except for very marked enlargement of the glands of the neck, which was relieved very promptly by the use of an ice-collar. On March 5th a well-defined erysipelatous swelling appeared over the left side of the face, about the temples and malar bones, and gradually extended over the entire face and part of the scalp. The eyes were completely closed, and the lips very much swollen. The mouth was very painful, being covered with sores to such an extent that it was impossible for the tongue to be protruded, and it was impossible for food to be taken. The throat was very dry, and a spray of listerine was used as mouth-wash. The ordinary treatment for typhoid fever was at once withdrawn, and the patient was put on thirty drops of the tincture of chloride of iron, three times a day. Under this treatment she improved, and by March 16th all inflammation had entirely disappeared, leaving only some swelling, which in the course of the next two weeks entirely passed away. The patient during this time continued to manifest symptoms of typhoid fever, and was unable to leave her bed on account of this disease for three weeks after the erysipelas had disappeared. Total recovery eventually took place.

The second case was that of A. E., a female aged twenty years, who was admitted to my wards with all the early symptoms of enteric fever, which developed into a moderately severe attack, but was without any extraordinarily severe symptoms. It was estimated that at the time the erysipelas developed she was in the

third week of the typhoid fever. At the onset of the erysipelas there was a chill, followed by a rise of temperature of 2°, and followed, after the use of a cold bath, by a fall to the temperature-course previously pursued. The erysipelas began about the bridge of the nose and extended rapidly over the entire face back to the ears and to the margin of the hair, whence it ceased to spread. The eyes were closed and the lips much swollen. An examination of the serum withdrawn by a lancet showed the characteristic streptococci of erysipelas. Under the use of large doses of tincture of the chloride of iron and an application of ichthyol ointment, recovery rapidly took place. The mouth was unusually foul and dry, but no delirium was present. We could not notice that the complication in any way increased the gravity of the case.

The third case is as follows: A woman, aged nineteen years, a Swede, was admitted in the early stages of typhoid, which ran a mild course, devoid of delirium or any symptoms of importance, except that on an afternoon, about the middle of the third week of her illness, she developed a sudden rise of temperature to 104°, followed at once, on the use of cold bathing, by a fall to 98°, with loss of the pulse at both wrists. As a precautionary measure, she was treated as if suffering from intestinal hemorrhage, and soon rallied, developing during the next twelve hours a typical patch of erysipelas on the right side of the nose and over the malar bone. There was no further disturbance of the typhoid temperature, and the disease remained limited to that side of the face. The patient was treated with iron and ichthyol.

By far the most exhaustive study which we have found concerning erysipelas as a complication of typhoid fever is that of Gerente.[1] According to this authority, the complication comes on in one of every sixty-one cases, which would give a much higher percentage than that of Liebermeister or Griesinger. Gerente states that females are more commonly affected than males, which is a curious fact, because males are more exposed and more frequently have typhoid fever. In regard to the period of the disease

[1] Thèse de l'École de Médecine, 1883–84, t. i.

at which erysipelas, as a rule, appears, Gerente states that it is generally after the twenty-first day, and he also believes that some epidemics of typhoid are peculiarly liable to this complication. The following conclusions of Gerente, however, embody most of his statements :

Eysipelas of the face is rarely met with during the course of typhoid fever. I have found it in 64 out of 3910 cases, which is about 1 to 61. These figures are derived from the following statistics :

	Typhoid fever cases.	Erysipelas. cases.
Chomel	130	4
Louis	134	3
Forget	92	1
Jenner	65	2
De Larroque	105	4
Zuelzer	84	3
Liebermeister	1420	10
Zuccarini	480	18
Griesinger	500	10
Murchison[1]	900	9
Total	3910	64

Outside of the question of contagion, it appears to be most frequent in the grave, adynamic forms of typhoid, and in those of long duration ; it appears to be most frequent in lymphatic subjects.

While observed at all the stages of typhoid fever, erysipelas shows itself especially and almost exclusively during the last period and during convalescence.

Under these circumstances erysipelas produces a marked amelioration in the general as well as in the local symptoms.

The appearance of facial erysipelas in the course of typhoid fever is of grave prognosis (sixteen deaths out of thirty-six cases) ; this gravity lies less in the erysipelas, which most frequently is benign in itself, than in the poor general condition of the patient, the secondary infection being an indication of this condition.

The complication consists in a simple coincidence favored by debility, the result of the primary and principal disease.

[1] The number of Murchison's cases is not strictly correct.

We think the statement that erysipelas seriously influences the prognosis in all cases too sweeping. Thus, there are cases on record in which the onset of the acute disease has not in any way retarded convalescence. If the disease becomes phlegmonous the prognosis is, of course, very grave; but if the inflammation is capable of undergoing resolution the prognosis is good.

The question as to the path by which contagion finds entrance has been much discussed, but the opinion of Griesinger is generally accepted. He believes that the germs gain entrance by means of the inflammation of the frontal or sphenoidal sinuses, and also when ulceration of the buccal mucous membrane exists. Zeulzer also points out that in his own cases and in those of Zuccarini the erysipelas started in the stomatitic spots and ulcerations in the mouth.

In all our cases the patients complained very much, both before and after the attack of erysipelas, of the soreness of their mouths. The following cases which have been reported in addition to the three of Gerente are interesting:

Armieux[1] reports the case of a soldier in whom typhoid symptoms set in on September 18, 1881, with pain in the head, vertigo, abdominal tenderness, pain in the right iliac fossa, and an elevated temperature. On October 4th a complication arose in an otorrhœa which, by the 22d, was growing steadily worse, so that the patient's condition was critical. Now facial erysipelas made its appearance, beginning in the auditory canal. Early in November osteitis of the humerus set in, and the patient died on November 9th.

Thielman[2] reports the case of a man, aged thirty years, brought into the hospital in an unconscious condition. The right ear, eyelids, nose, greater part of the face and forehead were covered with an erysipelatous eruption. The tongue was dry and brown; there was pain in the ileo-cæcal region, and the liver was painful and enlarged. The fever was recognized as typhoid, and the patient put upon calomel. The patient was in a delirious condition, but

[1] Rev. Méd. de Toulouse, 1875, ix. 42.

[2] Med. Jahresbuch v. Peter-Paul Hospital in St. Petersburg (1840, 1841), 142, 147.

on the following day there was a slight remission, and he became partly conscious. The erysipelas was seen to be spreading further over the face, but leaving its original seat. There was delirium the following night and semi-consciousness. Desquamation set in on the right side of the face, the eruption extending on the left. The pulse grew stronger, but the tongue was still brown in the centre. The patient was noticed to be troubled with occasional cough, and the respirations were somewhat more frequent. Examination showed a hypostatic congestion of the lungs. The condition became critical, but was relieved, and the patient gradually improved, being dismissed as cured on the thirty-fifth day after admission.

M. Berthoud[1] reports the case of a soldier who had typhoid fever of a meningeal type. The typhoid fever was declining, but convalescence was tardy, and his general condition was unsatisfactory. At this time the scrotum became tumefied and red, the redness spreading to the inguinal regions, while the general condition became very poor. The scrotum was triple its natural size, red, moderately warm, tender, not very painful, but œdematous, the redness extending to the right and left inguinal regions as far as the anterior superior spinous process, and also to the internal aspect of the thigh. The skin in these parts was swollen but soft, and the color persisted on pressure. On the next day there was no amelioration of the symptoms, but a very small area of necrosis appeared on the scrotum, which was treated by the application of the cautery. On the following day the necrosis seemed to be arrested and the scrotum reduced in size. The general condition, however, remained alarming. Six days later the patient died, after a subdelirium of four hours. The autopsy showed that the iliac and renal veins were involved in a plastic and suppurative inflammation, a case of erysipelas in the veins. The conclusion reached is that the redness of the skin and infiltration were due purely to mechanical causes, viz., the stagnation of the blood.

Freudenberger[2] has recorded two cases, in one of which erysip-

[1] Gaz. des Hôp. de Paris, 1848, vol. v. p. 29.

[2] Aertzl. Intelligenzblatt, München., 1880, xxvii. p. 37.

elas appeared suddenly on both ears in the course of typhoid fever, without unfavorable symptoms. On the following day a chill and rapid advance of the disease took place. The typhoid fever was now considered as declining, but the prognosis grave, because of the erysipelas. In the second case facial erysipelas suddenly appeared during convalescence from typhoid fever, although the temperature was already quite low. The fever became high again, but was easily influenced by antipyretics. The pulse was 140.

Potain[1] reports a case of erysipelas coming on during convalescence from typhoid fever, which was accompanied by a severe chill and fever. The erysipelas began in the pharynx and palate, and did not affect the tonsils. On the next day the inflammation appeared at the corners of the mouth and on the face.

Finally, Martinez[2] reports the following cases: A girl, twenty years of age, belonging to the lower class, of lymphatic temperament, with very irregular menstruation, which was often almost absent, was taken ill with typhoid fever. The symptoms were obscure at the onset of the disease, but the most prominent manifestation was an erysipelatous inflammation of foot and leg. On the fourth day the erysipelas was marked; there was great fever, cephalalgia, and other typhoid symptoms, such as weakness, gurgling in the right iliac fossa, dryness and tremblings of the tongue, sordes on the teeth, great stupor, delirium, and a frequent and small pulse. Death took place after some days.

Whether the erysipelatous trouble had anything to do with the causation of the typhoid symptoms or not, Martinez does not state, but he mentions the case of another woman in whom an extensive erysipelatous inflammation of the face and scalp produced cerebral symptoms, fever, etc., but they were not so pronounced as to be confounded with those caused by true typhoid fever, as in the present instance. In this case the patient recovered.

It is an interesting fact in this connection that Silvestrini[3] has

[1] Erysipèle de la Face Consecutif à la Fièvre Typhoïde. Gaz. des Hôp. de Paris, 1880, liii. p. 1106.

[2] La Espana Médica, Madrid, March 1, 1860, p. 135.

[3] La Riforma Medica, 1894, 196, 197.

met with two cases of facial erysipelas in typhoid fever, in which the inflammation was found to be due not to streptococci but solely to the bacillus of Eberth. He asserts that Klebs and Reiner have met with similar cases.

Very often in the last week of defervescence and in convalescence the patient suffers from colliquative sweating of a marked type. It has seemed to the writer that in these cases the flow of sweat was an effort at elimination.

Taupin[1] tells us, in an article written as long ago as 1839, that in children it is common to meet during convalescence with very abundant sweating of the upper part of the body, while the lower parts remained dry, and that children convalescing from typhoid fever might be attacked by an eruptive fever. He also speaks of cases attacked by scarlet fever, smallpox, and measles, due, in all probability, to the lack of isolation in fever wards in those days.

Amitrano[2] has recorded a case of typhoid fever which, during convalescence, developed the scarlatiniform rash which desquamated. Marked meningeal symptoms developed after the fever subsided, and after desquamation was completed a second erythema of the skin appeared, which was also followed by desquamation. This case, perhaps, belongs to the class of dermatitis exfoliativa. (See last chapter for a discussion of typhoid fever complicated by eruptive diseases.)

Profuse desquamation of the skin is frequently met with in patients convalescing from typhoid fever. The writer has seen this again and again, and Comby[3] speaks of it as a state met with in the convalescent period in children.

Coulon[4] has recorded a case of typhoid fever in a child of ten and a half years, in which there was general desquamation of the skin during convalescence; previous to that there had been no eruption upon the skin. On the other hand, it is noteworthy that there had been sore-throat, albuminuria, and œdema, so the case

[1] Journal des Connaissance Medico-Chirurgicale, 1839, No. 7.
[2] La Riforma Medica, 1896, No. 146.
[3] Gazette des Hôpitaux, 1896, No. 39.
[4] La Médicale Enfantile, January, 1895.

may have been one of scarlet fever complicating typhoid, and without the ordinary rash.

A somewhat unusual lesion of the skin, resulting from typhoid fever, is the development of lineæ albicantes. Cases of this kind have been reported by Troisier,[1] and Manouvriez and Bouchard have also recorded such instances. It is stated that they occur most frequently in children and young adults. Bucquoy notes that in boys these whitish lines have no special area of distribution, but in girls the breasts and crests of the ileum are the places where they usually appear. Barié has reported the case of a girl of seventeen years, in whom these lines appeared over the knuckle-joints of each hand.

A somewhat similar condition, due to localized atrophy of the skin, is recorded by Bradshaw.[2] In his case a girl of thirteen years, who suffered from typhoid fever followed by relapse, and again by a second relapse, finally developed during convalescence upon the inner surface of the lower third of the thigh a number of horizontal markings, some of which partially surrounded the limb; they were about one-half inch in width, regular in contour, and almost exactly alike on both legs. A similar condition has been described by Wilkes.[3]

A very rare condition coming on during convalescence in typhoid fever, is reported by Leudet,[4] namely, the condition of painful œdema of the thorax. Pain was first felt in the neighborhood of the thyroid gland, then in the shoulder-blade; later a circumscribed œdema of the left side of the thorax developed, which was not reddened, but was painful to the touch. There was no fever and no albuminuria. The condition lasted for four days in its fully developed stage, but had disappeared entirely by the twelfth day.

The Thyroid Gland. The thyroid gland may undergo suppuration as a result of typhoid fever, as it may in other infectious

[1] Bulletin et Memoire de la Société Médicale des Hôpitaux, 1889, No. 12.

[2] Bristol Medico-Chirurgical Journal, July, 1889.

[3] Guy's Hospital Reports, 1861.

[4] La Normandie Médicale, October 1, 1891.

processes. Thus Pinchaud[1] has recorded such a complication of convalescence, and Forgue,[2] a Major in the French Army, has made a contribution on this condition. Other observers have recorded a similar state complicating the other infectious diseases, and the view is generally held that the gland becomes infected from the entrance of the bacillus into the blood, by which it is carried to the thyroid gland. The most recent paper on this topic with which I am acquainted is that of Testevin,[3] a Major in the French Army, who under the title of "Thyroidite Infectieuse Suppurée," discusses the literature of the subject. From his paper it is evident that of all the infectious diseases, typhoid fever is the one which most commonly causes these lesions in this gland, and further, that it is emphatically a consecutive or secondary manifestation chiefly met with in convalescence. In very rare instances the thyroiditis develops with the onset, as set forth by Tavel[4] and Laveran.[5]

Finally, it is a noteworthy fact that Chantemesse[6] has found the bacillus of Eberth in the pus of the thyroid gland.

A case of suppuration of the right lobe of a goitrous thyroid gland has been recorded by Spirig,[7] in a woman, twenty-two years of age. This complication arose after five weeks of typhoid fever, when the disease was on the decline; both the bacillus of Eberth and the staphylococcus were found in the pus.

Joints. Articular lesions complicating convalescence from typhoid fever may be due to direct infection with the specific bacillus, which is rare, or to infection by other organisms. This question is ably considered in Dr. Keen's monograph, already quoted, and does not need to be discussed at this point for this reason.

[1] Des Thyroidités dans la Convalescence de la Fièvre Typhoïde, Paris, 1881.

[2] Contribution à l'Étude de la Thyroidite Typique. Arch. de Méd. et de Phar. Milit., 1886, 1. vii.

[3] Ibid., February, 1899, p. 126.

[4] Ueber die Etiologie der Strumitis, ein Beiträge zur Lehre von den Hematogenen Infectionen, Bâle, 1892.

[5] Revue de Chirurgie, Septembre, 1890, No. 29.

[6] Art. Fièvre Typhoïde in Traité de Méd. de Bouchard et Charcot, 1891, 768.

[7] Correspondenzblatt für Schweizer Aerzte, February 1, 1892.

Robin and Leredde[1] have, however, called attention to the interesting fact that acute articular inflammation is sometimes met with in typhoid fever, and believe it to be rheumatic in some cases. On the other hand, in the great majority of instances the joint affection is not due to acute articular rheumatism, but it is simply an evidence of the septic process associated with the typhoid fever. Great care should be exercised by the physician that mere articular inflammation does not mislead him in an erroneous diagnosis.

As is well known, dislocations have been recorded in considerable number as having occurred during the progress of typhoid fever and in acute rheumatism. In the first of these diseases the displacement of the bone has occurred in the earlier days of convalescence, when the patient has been so feeble that it has seemed as if the accident was due to the relaxation of the coverings of the joint and its associated muscles, with the result that the bone has easily slipped out of place, and in nearly all these cases there has been no evidence whatever of any local difficulty prior to luxation. On the other hand, in acute articular rheumatism where dislocation has taken place there has nearly always been a history of arthritic difficulty prior to the accident, and instead of the dislocation producing pain of a moderate degree, as it has done in convalescence from typhoid fever, the occurrence of the displacement has been followed by great relief from pain, owing to the overcoming of the vicious attitude which has been maintained by the limb. The cases of scarlet fever in which this accident has occurred have belonged rather to the typhoid class, in that the dislocation has taken place without much pain, and, therefore, without attracting great attention to its presence. As long ago as 1882 Rawden reported in the *Liverpool Medico-Chirurgical Journal*, an instance of dislocation following typhoid fever, in which, having excised the head of the bone, he found it practically normal, even the cartilage being healthy, excepting for a little absorption at its periphery; while, on the other hand, Adams, in a case of rheumatic dislocation of the hip, found the capsular ligament

[1] Archives Générales de Médecine, September, 1894.

ruptured and the torn margins of the rent closely embracing the neck of the bone. While it is true that unobtrusive monarticular synovitis with effusion may take place in convalescent patients, the literature of the subject does not reveal the fact that post-typhoidal dislocations have usually been due to this condition, and Collier believes that degenerative changes similar to those seen in muscular fibres result in softening of the ligaments and of their attachment to the bones. The possibility of recurrence of the dislocation under such circumstances is great, and the prognosis as to the correct use of the limb must be made with caution, since some cases seem to become entirely well, while others never get rid of a certain amount of anchylosis or shortening.

In this connection it may be a matter of interest to note that the case of typhoid fever under my care in the wards of the Jefferson Medical College Hospital in the early part of 1897, to which reference is made in Keen's essay, page 97, has been seen by me in March, 1899. She is able to walk without the aid of a crutch, but the knee is permanently anchylosed. It will be remembered that aspiration of this knee-joint obtained fluid which was perfectly sterile. A much more interesting point in connection with the case, from a prognostic point of view for other cases, is that the anchylosis in marked flexion, which Dr. Keen thought would require operative treatment later on, has been gradually overcome, so that shortening in the anchylosed limb is very slight.

CHAPTER V.

THE CONDITIONS WHICH APE TYPHOID FEVER.

These conditions are quite numerous. The following is a list of the more common of these conditions: Malarial fever, appendicitis, sepsis, pneumonia with great asthenia, tuberculosis, particularly of the abdominal contents; ileo-colitis, ulcerative or septic endocarditis, and cerebro-spinal meningitis.

With the important question of the diagnosis from malarial fever I have already dealt in the chapter on the Well-developed Stage of the Disease. The important facts for the physician to remember are that the infection by the bacillus of Eberth and that by the parasite of malarial fever may pursue a course in each case almost identical with the other, and that in such cases a differential diagnosis is to be made chiefly by means of the Widal test on the one hand and a search for the malarial organism on the other. It is also to be recalled that the quinine test is not of great negative value, and that its persistent use in a malarial case may simply make the microscopic diagnosis impossible. On the other hand, the use of quinine for several days when without result should not be persisted in, since the case under these circumstances is probably not due to malaria. Speaking of this therapeutic test, Dock well says: "In a case resembling typhoid fever, but really malarial, the microscope is essential to good practice. Without it, quinine may again be used; but if the temperature does not fall to or near normal, with relief to the other symptoms, it is better to stop quinine altogether. Only when microscopical evidence of malaria is present should the drug be pushed after the third day. It is necessary to add that while symptoms persist the patient should be treated as though he had typhoid fever. So erroneously is the so-called therapeutic test conceived, that I have known of patients

taking quinine in doses of forty grains a day for three weeks, in order to determine the presence of malaria, each fall of one or two degrees of temperature being looked on as proof of a specific effect. I am well aware that some look on massive doses of quinine as useful in typhoid fever, but considerable observation has convinced me of the opposite view."

With these views, particularly those of the last sentence, the writer is in entire accord. The facts already well emphasized in this essay, that severe chills, rigors, and sweats may appear in many cases of typhoid fever entirely devoid of any touch of malaria, proves that all these signs are not proof of malarial infection. In confirmation of these views we find the interesting report of Ewing,[1] made after his able studies among soldiers of the Spanish-American war at Montauk Point, in which he says:

"The reason why the blood was examined in 159 cases of typhoid fever, was the intermittent character of the fever, which was exhibited in patients both with and without malarial antecedents. In no case of undoubted and established typhoid fever were malarial parasites found in the blood in connection with any of these sudden rises of temperature, but only at the onset of the disease or during the convalescence.

"On the other hand, many patients whose blood contained numerous parasites were seen in the 'typhoid state,' but there were always some essential symptoms lacking to confirm the diagnosis of typhoid fever, while the subsequent course of the disease demonstrated the purely malarial character of the fever.

"These patients might suffer from epistaxis, hæmatemesis, bloody stools, tympanites, a few rose spots, though oftener herpes, diarrhœa and delirium, and in some a partial Widal reaction was obtained. But the intestinal symptoms were inconstant or referable to dysentery or simple diarrhœa, from which many of the malarial cases suffered, and these patients never showed subsultus or cracked tongues, and they did not die, or, if they did, dysentery and malaria were demonstrated at or before autopsy."

[1] New York Medical Journal, February 4, 1899.

Again, he says: "It is possible that some of these patients suffered from both active malaria and typhoid fever, but there were no positive indications that the latter infection was present. In the cases that came to autopsy there was never any doubt of the nature of the disease. It was either typhoid fever or malaria, but never both, although microscopical evidence of dormant malarial infection was found in at least two cases of typhoid fever.

"In short, in spite of very painstaking efforts, the attempt to find a case of typhoid fever and active malaria progressing simultaneously was unsuccessful.

"From a study of this group of cases it is concluded:

"1. That typhoid fever is to a large extent incompatible with active malarial fever, and that during the course of the former the latter infection is usually suppressed.

"2. That the presence of old malarial infection may alter the course of typhoid fever through the anæmia, but that active sporulation of the malarial parasite very rarely occurs during the course of established typhoid fever.

"3. On the other hand, since malarial paroxysms often reappear during convalescence, a scanty growth of the parasite must often persist during the course of typhoid fever, and it is possible that some of the irregularities of temperature observed in these cases are referable to this partly suppressed growth.

"4. That the anatomical evidence of a post-mortem examination is much needed to demonstrate the existence of typhoid fever in cases showing active malarial paroxysms."

A valuable paper upon the relations of typhoid fever to malarial infection was published some years ago by Gilman Thompson,[1] in which he reached results identical with those just stated, namely, that the fever of typhoid is apt to run its course, and that malarial manifestations then succeeds it.

Sepsis and *appendicitis* may somewhat closely resemble typhoid fever if the latter affection is insidious and there is pus present which produces toxæmia. Whatever the cause of the sepsis may

[1] American Journal of the Medical Science, August, 1894.

be, the loss of flesh, dry tongue, delirium, low-grade bronchitis, badly nourished skin, and diarrhœa may cause the patient to be most typhoidal in appearance, yet in all such cases we should seek for a possible purulent focus. The absence of the Widal reaction and the presence of leucocytosis should rouse our suspicions greatly, and it is not to be forgotten that the presence of pus deep in the pelvis or in the neighborhood of the kidney may not be readily discovered, and only the development of fluctuation or the rupture of the abscess will force the physician to reverse his diagnosis of typhoid fever. On the other hand, as already pointed out, purulent formations may occur in typhoid fever.

Similar facts make us suspect and search for signs and causes of ulcerative endocarditis in such cases.

The fact that tuberculosis may simulate typhoid fever, and that cerebro-spinal meningitis may likewise do so, has already been discussed in the foregoing pages, but it is not out of place to point out that four types of tuberculosis are particularly apt to produce misleading symptoms. In tubercular meningitis the febrile movement is rarely as high as in typhoid fever with associated meningeal symptoms ; the abdomen is usually scaphoid instead of tympanitic, and the persistent vomiting of the former disease is comparatively rarely met with in the latter. An ocular examination may reveal optic neuritis in tubercular meningitis, or paralysis of the muscles of the eyeball, causing squint.

So, too, in acute general miliary tuberculosis, the previous history of the patient as to gradual failure of health, and cough, the moderate fever, and the rigors and sweats point to the presence of tuberculosis rather than enteric fever. Further, there will be in some cases marked physical signs of widespread involvement of the lungs in tuberculosis which will be absent in typhoid fever. It is to be recalled, however, that a roseolous rash may develop in both affections, and that diarrhœa and a dry, brown tongue may mislead the careless very readily. Even intestinal hemorrhage may occur in miliary tuberculosis.[1]

[1] Senator. Charité Annalen, 1892, vol. xvii. p. 272.

Tubercular peritonitis may also cause typhoid symptoms, but as the disease progresses the localization of the abdominal symptoms and, finally, the development of tumor masses or enlargement of the mesenteric glands, can be felt on deep palpation, or, in other cases, the development of ascites makes the diagnosis clear.

Finally, it is not necessary for the rather rare disease, general miliary tuberculosis, to be present to make the diagnosis obscure. Some time since I saw in consultation a man of thirty years, who had had for four weeks persistent fever, some cough, diarrhœa, mild delirium, gradual loss of flesh, and a heavily coated tongue, with sordes. To the attending physician who had made a diagnosis of enteric fever at the start, nothing had occurred to make him change his views, but the appearance of the patient made me suspicious of tuberculosis, and a careful examination of his chest revealed well-advanced tuberculosis of the lungs, the real cause of his illness.

Girandau[1] has recorded a case in which a young man suffered from enteric fever, and then speedily developed tuberculous disease of the intestines. Two weeks after the recovery from enteric fever, the patient became ill a second time with diarrhœa, fever, and abdominal pain, and marked wasting. At the autopsy two sets of lesions were found, typhoid lesions side by side with tubercular foci. No traces of old pulmonary lesions or a primary lesion elsewhere were to be found.

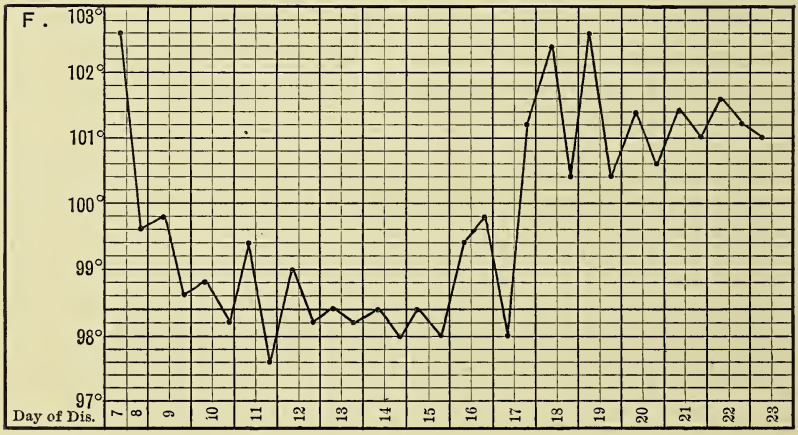
An interesting case illustrating how difficult the diagnosis of typhoid fever may be in its earlier stages has recently been under my care:

This woman was taken ill some days before I saw her with chilliness, fever, and languor, and with a further history that she had been suffering for a number of months with somewhat similar sensations, without the fever, and had been losing flesh; during this time she had had constipation alternating with diarrhœa and abdominal pain. When first seen her temperature was 103°, her appearance was distinctly that of a typhoid patient; but, as is seen

[1] Revue de Médicine, 1884, p. 564.

in the accompanying chart, her temperature speedily fell to normal, only one sponge bath being required after she came under observation. An examination of her abdomen at this time revealed the fact that it was slightly protruding, and that the abdominal wall was so thin that the coils of intestine could be readily seen projecting through it. In the neighborhood of the umbilicus there was a sense of increased tenderness on deep palpation, and the resistance made one suspect the possibility of there being present a tubercular peritonitis which had caused an exudation, binding the intestines together in a mass. About McBurney's point there was

Fig. 21.



A case of typhoid fever preceded by appendicitis (?), or by a primary attack of typhoid fever.

very distinct tenderness on palpation, and deep palpation produced severe pain. In view of her history, her emaciation, and the symptoms detailed, I was inclined to consider the case one of tubercular peritonitis, or else one of appendicitis of the subacute or chronic character, with a tendency to exacerbations. In this opinion Professor Keen agreed with me, and it was arranged that Professor Keen should perform an abdominal section for the purpose of removing the appendix, if it alone was the cause of the difficulty, or of relieving her tubercular peritonitis through the well-known beneficial effects of abdominal section. On the day on which she

was to be operated upon, her temperature having been normal for a number of days, and her general condition having steadily improved under treatment designed to prepare her system for operation, she developed marked languor and malaise and febrile movement, which is shown in the accompanying chart (Fig. 21), and three days later developed typical rose rash of typhoid fever, her blood giving the positive Widal reaction simultaneously. The questions which naturally arise in regard to this case are: Did the woman suffer primarily from appendicitis, or from tubercular peritonitis, or did she come under my care at the end of a mild primary attack of typhoid fever after which she had a relapse, or, again, is it possible that suffering from a mild chronic intestinal catarrh, she received typhoid infection just prior to her entering the ward, thereby superimposing typhoid fever upon the condition present when we first saw her? Because of her ultimate complete recovery I am inclined to believe that the primary fever could not have been due to tubercular peritonitis.

Another interesting case, illustrating how difficult these differential diagnoses may be, is reported by Dreschfeld in Allbutt's *System of Medicine*, in which three members of one family that had lived in a cellar which had been under water at the time of an extensive flood were attacked with a fever. Their symptoms closely resembled those of enteric fever, and one of them presented on the third day after admission marked roseolar spots, and had slight intestinal hemorrhage on the fifth day. The temperature showed marked exacerbations, and the patient died from exhaustion on the fourteenth day after admission, or about the seventeenth day of the fever. The post-mortem examination revealed the intestines apparently healthy. Dreschfeld says he can quote similar cases. He does not state what he believed this illness to be due to, but from the context he evidently regarded it as septic, although the absence of intestinal lesions, as I have already stated, does not exclude enteric fever.

Leu[1] has reported a case of puerperal septicæmia which was

[1] Charité Annalen, 1891, vol. xvi. p. 315.

almost indistinguishable from typhoid fever, for the patient had a rose rash, tympanites, enlarged spleen, intestinal infection, and the pyrexial curve, which is characteristic. The fact that puerperal septicæmia is fatal within a few days; that there is a local focus of the disease, and that such a disease would not present the Widal reaction, aids us in making a differential diagnosis.

Another condition which may closely simulate enteric fever is the gastro-intestinal form of epidemic influenza, for in this condition we have enlargement of the spleen, diarrhœa, tympanites, gurgling, slight evidences of bronchial irritation, and very rarely, indeed, a suspicious roseolar rash. On the other hand, it is perfectly possible for enteric fever and influenza to occur simultaneously in the same patient.

Under the name of *mountain fever*, a febrile disease occurring in the great highlands which occupy the middle portion of the United States, has been described by a number of authors. Some of these writers have been strongly of the opinion that mountain fever is a distinct entity, while others have gone so far as to assert that it is an irregular manifestation of malarial poisoning, and still others that it is a modified form of typhoid fever.

As a matter of fact, we may state positively at this time that true mountain fever is in all cases nothing more than a greatly modified or altered type of typhoid infection. As has already been pointed out in this essay a number of times, typhoid fever is a disease which varies greatly in its symptomatology and course, and does not, in many instances, follow the classical descriptions of it which we are accustomed to find in the text-books.

One of the most conclusive and interesting papers dealing with this matter which is to be found in recent literature, is that of Raymond, who, as post surgeon at one of the United States Army stations in the West, has contributed to the *American Journal of the Medical Sciences*, 1898, vol. cxv., an exhaustive paper upon this subject.

Quinine administered to these cases, in full doses, failed to exercise any beneficial effect; prophylactic measures, which are ordinarily successful in the control of the typhoid epidemic, at once

checked the disease, and a comparison of many of the symptoms manifested with those met with in irregular forms of typhoid fever still further indorse the view we have already expressed in regard to the unity of these two diseases.

These views in regard to mountain fever are also supported by the paper of Work,[1] who tells us that eighteen out of fifty cases of mountain fever, so called, had rose spots, and that in five fatal cases the intestinal lesions of the fever were found.

The differential diagnosis of typhoid fever in children from the other exanthemata is made as follows: From scarlet fever by the pain in the back, the excited nervous system, the eruption on the second day, and the absence of pain in the abdomen, and the stupor of enteric fever. There is usually in scarlet fever, too, great sore-throat. From measles we are apt to have greater bronchial catarrh, at least at first; coryza, which is very rare in typhoid fever, and an early eruption. From entero-colitis we distinguish enteric fever by the absence of delirium or stupor in this affection, and the character of the diarrhœa, as well as the greater abdominal tenderness. The value of the Widal test in these cases is never to be forgotten.

[1] Medical News, April 8, 1894.

CHAPTER VI.

DURATION AND IMMUNITY TO SECOND ATTACKS.

The duration of typhoid fever varies greatly in different individuals, and still more so in different epidemics, depending upon the vital resistance of the patient and the virulency of the infection. It may, however, be asserted that the average period of fever is twenty-one days, although wide variations from this may occur, the duration being much less or much greater, as already pointed out.

Murchison states the mean duration in seventy-five cases to be a fraction more than twenty-four days. Flint states from going to bed to normal temperature sixteen days, with a maximum of twenty-eight days and a minimum of five days. The longest case seen by Flint was fifty-eight days.

Of forty-five of Flint's fatal cases the duration was a fraction more than fourteen days. Murchison tells us that the mean stay in the hospital of 500 cases which recovered was 31.24 days; of 100 fatal cases, 16.52 days, while the average duration of illness before admission of the 600 cases was 10.78 days. Again, Murchison tells us that the pyrexia, as a rule, lasts at least three weeks, and the ordinary duration of enteric fever is from three to four weeks. Of 200 cases which recovered, and in which he was able to fix the commencement with tolerable certainty, the duration was: 10 to 14 days in 7 cases; 15 to 21 days in 49 cases; 2 to 28 days in 111 cases; 29 to 35 days in 33 cases.

The mean duration of the 200 cases was 24.3 days, and the mean duration of 112 other cases, which were fatal, was 27.67 days.

The average duration of residence in St. Thomas' Hospital, London, in 1894, 1895, and 1896, was from 43.1 to 51.8 days, and the average duration of fever from 14.3 to 16.73 days, although a great proportion of the patients were admitted in the first or second week.

In the Maidstone[1] epidemic, 8 per cent. lasted two weeks; 27 per cent., three weeks; 31 per cent., four weeks; 17 per cent., five weeks; 8 per cent., six weeks; 4.5 per cent., seven weeks; 84.5 per cent., eight weeks.

If we take the twenty-five cases admitted in the first week of the disease given in Wilson's table, we find that the average stay of these patients in the house was forty-one days ($40\frac{8}{9}$), and the average day of normal temperature the nineteenth. The average maximum temperature was 104.6° If the entire 108 cases given in his last table in his article are studied, we find that the average duration of the fever was in the cases admitted in the second week, 23.2 days; in the third week, 27.3 days, and the average stay in the house of the second-week cases, 40.8 days, and of the third-week cases, 38.8 days.

While the general average may be about twenty-one days, very much shorter periods have been seen and noted by every physician of experience, and very important classifications of cases have been made by Liebermeister and Jurgensen. The first of these clinicians speaks of the mildest cases as those in which the rectal temperature never or rarely rises above 103°, and the duration of fever does not exceed eight days. The mild cases do not have a rectal temperature above 104.8°, and the fever lasts sixteen days. The severe cases are those in which the rectal temperature rises above 105° and the fever ceases by the twenty-first day. Jurgensen considers all cases mild which have no fever after the tenth day, and those severe that have fever after this date; but this view hardly coincides with that of American physicians, who regard a fever ending by the twenty-first day as quite moderate, particularly if the fever does not exceed 104°.

There is one class of patients in which the febrile movement very commonly lasts but a week or two, namely, children. Henoch stated years ago that out of 80 cases seen by him there were 11 which lasted 7 to 10 days; 26 from 10 to 15 days; 16 from 15 to 20 days; 21 from 20 to 30 days, and 6 from 30 to 49 days.

[1] Poole. Guy's Hospital Reports, 1898. Wrongly labelled on cover, 1896.

Even in the cases lasting but a week or ten days there were roseola, enlargement of the spleen, and diarrhœa. In confirmation of this view, we have the more recent observations of Forchheimer, of Cincinnati, who found in an epidemic of this malady among children that the fever may terminate as early as the sixth day, and Janeway, of New York, remarks that it may end in ten days. It is evident, therefore, that the duration of typhoid fever in children is shorter than in adults, as a rule, as well as milder in the character of its manifestations, and that it is accompanied by less grave intestinal lesions.

Musser has recorded the case of typhoid fever in which, though there were no complications, the temperature did not reach normal until seventy-three days had elapsed.

In children convalescence is even more prolonged than it is in adults in some cases. As long ago as 1839, Taupin[1] emphasized this fact, stating that pallor, feebleness, and general debility are marked.

The question of the frequency of second attacks of typhoid fever is of interest. It is generally considered that an attack renders a patient at least partially immune to other attacks. Moore[2] has recorded a case of a man who suffered from typhoid fever at fifteen years and again at twenty-nine years, and finally from a relapse after this second attack, and Leidy[3] has reported a case of a boy who had an attack of enteric fever at sixteen years, a second attack six months later, a third at the age of thirty-four years, and this followed by four relapses, in the third of which he had intestinal hemorrhage, but recovery nevertheless occurred. During the winter of 1897–98 the writer had under his care a boy who was suffering from his third attack of typhoid fever, his first having occurred at nine years of age, the second at seventeen years, and the third at nineteen years. Death occurred from hemorrhage of the bowels. In 1626 cases Bey found only one which had a second attack.

[1] Journal des Connaissance Méd. Chirurgicale, July, 1839.

[2] Dublin Journal of Medical Science, April, 1893.

[3] International Medical Magazine, August, 1893.

CHAPTER VII.

THE MENTAL COMPLICATIONS.[1]

The mental complications of typhoid fever resemble in a general way the mental disorders resulting from other infectious diseases. They occur by preference in patients in whom there is present a neurotic heredity or who have been subjected, previous to infection, to overwork, loss of sleep, anxiety, or other exhausting nervous strains. Hereditary factors—functional neuroses and insanities—appear to be present in about half the cases. It cannot be claimed, however, that the other predisposing causes possess much etiological value, as mental complications frequently occur in individuals in which these factors have been absent. Sex appears not to exercise any predisposing influence, males and females being affected in about equal number. Age, also, is not a determining factor. It is, however, somewhat significant that typhoid fever attacks by preference individuals of an age at which mental disorders are very prone to occur, namely, youth and early adult life. Notwithstanding, mental diseases of typhoid origin of sufficient severity to demand asylum treatment do not appear to be as frequent as this coincidence would suggest. Thus Nasse reported 43 cases among 2000 hospital admissions; Schlager, 22 cases in 500; Christian, 11 in 2000, while Pilgrim found only 13 cases in over 6000 admissions. We should remember, however, that hospital statistics cannot be regarded as in any sense representing the real frequency of these disorders. First, a large number of cases do not necessitate commitment, and, secondly, in hospital admissions the etiological relation with typhoid fever is not always brought to the attention of the asylum physicians.

[1] By F. X. Dercum, M.D., Clinical Professor of Diseases of the Nervous System in the Jefferson Medical College.

The occurrence of typhoid insanities appears to depend, among other things, on the character of the individual epidemic; they occur more frequently in some epidemics than in others. Among special factors it is not improbable that constipation may be a predisposing cause, by favoring the retention and absorption of poisons.

The mental disturbances of typhoid fever are separable into three groups: First, those which develop during the prodromal or initial period; secondly, those which arise during the continuance of the fever, and, thirdly, those which occur during or subsequent to convalescence.

The affections occurring during the prodromal period cannot be definitely separated from those occurring during the initial period of the fever, inasmuch as cases beginning in the prodromal period may persist after fever has made its appearance. They manifest themselves in one of two forms: First, a form in which mental depression or mental excitement is the leading feature, and, secondly, a form in which the symptoms are those of an acute delirium. The first is represented by a class to which Campbell[1] calls attention. They begin in the prodromal period, and are especially prone to occur when this period is protracted. They appear to be directly related to the malaise and degree of nervous prostration. They are not infrequently met with in those cases in which the fever is slow in making its appearance or does not become pronounced until a considerable time has elasped. They are characterized by mental depression, less frequently by mental excitement, associated with disordered mental action—probably confusion, with some hallucinations. It is not surprising that the mental condition may entirely mask the underlying disease. The symptoms may be so pronounced as to lead to the commitment of the patient to the asylum, the nature of the case not becoming evident until later. It is extremely probable that in such cases there is a marked hereditary tendency to insanity, and that the depression of the prodromal period of the fever merely acts as an exciting cause. It should be added that these cases are quite rare. We

[1] Campbell, Colin M. Dict. of Psycholog. Med., vol. i. p. 506.

should, however, remember that if a given case is obscure in its origin, if the mental depression has developed in a manner more rapidly than that seen in melancholia, and if it is otherwise atypical, the commitment should, if possible, be delayed and the case be kept under observation for some days. The occurrence of this form also shows how important it is to make a thorough *physical* examination of the patient.

In the second form of mental disorder of the prodromal or initial period, we have present, as already stated, the symptoms of an acute delirium. This delirium is characterized by profound mental obtusion, confusion, and hallucinations, which are often terrifying in character. There are manifestations of great fear and often impulses to violent acts. In this form violent assault upon the person, murder, or suicide may occur. It may, indeed, in rare cases attain the violence of typho-mania[1] (delirium grave). (See chapter on onset.) While the delirium is usually accompanied by terrible hallucinations, the patient seeing frightful objects and hearing terrifying sounds, it is under rare circumstances associated with expansive ideas. Kirn[2] describes a case in which instead of depression there was present delirium of grandeur, only, however, to be followed by depression later on.

The acute delirium of the initial period is to be looked upon as among the unusual mental complications of typhoid fever. It appears to be present especially in certain epidemics, as, for instance, in that recorded by Blanc[3] as occurring among French troops in Tunis. Whether the delirium actually antedates the outbreak of fever is uncertain, inasmuch as accurate temperature studies are as yet lacking. It may, however, continue for some time after the fever has been established, and may merge into the ordinary fever delirium. In other cases, again, it disappears altogether as the height of the fever is reached. Many cases, however, die before the fever has fully developed. The existence of acute delirium in the prodromal or the initial period of typhoid

[1] Nasse. Allegemeine Zeitschr., f. Psych., 1870–71, p. 11.

[2] Ibid., vol. xxxix. p. 741.

[3] Schmidt's Jahrbücher, vol. ccxiv.

fever is always to be looked upon as of ill omen. According to Adler,[1] only one-third of the cases presenting this complication recover.

The mental complications occurring during the period of fever separate themselves into, first, the ordinary fever delirium; second, expansive or ambitious delirium, and, third, stupor or coma vigil. The fever delirium is ordinarily quiet in type, and, though at times associated with excitement, does not merit separate consideration here. The expansive or ambitious delirium, a rare form of complication, may be present during the entire course of the fever. More frequently it comes on after the fever has passed its height, and persists during the period of decline. In such cases the patient presents the picture of the delirium of grandeur. In a case observed by the writer the patient kept talking about his bags and vaults of gold, about his diamonds, fast horses, and other great worldly possessions. The delirium is not accompanied by marked excitement, and disappears with the defervescence of the fever.[2]

The stupor of typhoid fever, like the ordinary fever delirium, is so well known as not to merit description. It may come on as a gradual deepening of the initial apathy and hebetude of the disease, or may be a transition from the fever delirium. More rarely it is the outcome of an acute delirium of the initial period. Its occurrence at an early stage is always of grave significance. When arising during the period of decline it sometimes continues long after the fever has subsided.

The insanities which arise during or subsequent to convalescence are those which principally concern us here. They may arise during the subsidence of the fever, and may be merely a continuation of the confusion and delirium of the febrile stage; much more frequently they make their appearance after the fever has entirely disappeared.

[1] Allegemeine Zeitschr. f. Psych., vol. liii. p. 753.

[2] Cases have been reported by Delasiauve, Christian, Simon, and Liouville. Dict. of Psycholog. Med. vol. ii. p. 986.

Post-typhoid insanities may make their appearance in one or other of the following forms:

1. Acute delirium.
2. Confusional insanity, stuporous insanity.[1]
3. Cerebral asthenia, pseudo-dementia, pseudo-paresis.
4. Insanity with systematized delusions resembling paranoia.
5. True melancholia or true mania.

1. **Acute Delirium.** The acute delirium following typhoid fever is indistinguishable from the delirium of exhaustion following other infectious fevers, shock, trauma, or other profoundly debilitating causes. It is characterized by excessive mental confusion, increased rapidity in the flow of ideas, numerous and varied hallucinations, obtusion of the perceptive faculties to both internal and external impressions, and marked motor excitement. The onset is usually sudden, and frequently corresponds with the termination of the fever. It appears to coincide with the collapse which follows the disappearance of the fever in some cases. At other times a brief interval of a day or two characterized by insomnia and ominous restlessness precedes the outbreak. Consciousness becomes much obscured; the patient loses the proper recognition of his surroundings; he becomes illusional, everything seems strange and changed, and in addition he becomes hallucinatory to an extreme degree. The chairs and other objects of furniture are mistaken for strange shapes, persons, or animals. The individuals about his bed are no longer properly recognized; the pictures upon the walls, the curtains upon the windows, the rugs upon the floor all become animate objects. The hallucinations rival the illusions in their variety and number. They appear to consist especially of auditory and visual sensations. Voices call to him, strange persons, horrid creatures gesticulate, beckon, terrify him. It is not strange under these circumstances that he

[1] Kraepelin, Lehrbuch Psychiatrie, is one of the few systematic writers to fully appreciate the etiological relation of typhoid fever to these disorders. Paglians, Revue de Méd., 1894, xiv. 549 and 656, unfortunately misinterprets, as did the older writers, post-typhoid conditions attended by excitement or depression as mania or melancholia.

appears to have dreadful and depressive delusions. He believes that horrible punishments are to be meted out to him; that he is to be cut, to be stabbed, to be poisoned, that he has only a short time to live. No wonder that his struggles are often merely the outward expression of a frenzied fear. Very rarely the hallucinations and the delusions are of a pleasurable and expansive character, the patient showing by his demeanor, as well as by his speech, the pleasure that he feels. Sometimes he is distinctly erotic. Occasionally depressive and expansive mental states are present at different times in the same case.

The speech of the patient, in keeping with his disturbed mental condition, is for the most part fragmentary and confused, and the delusive ideas are difficult, if not impossible, to follow. Of course, the delusions themselves are fragmentary and unsystematized. The patient cries out or utters merely parts of sentences and phrases, and when the condition is fully established his words may be entirely incoherent or consist of senseless alliterations. At other times he talks excitedly, loudly, pathetically, or whispers, gesticulates, and makes grimaces. It is generally impossible to obtain a rational answer to a question, though sometimes during a momentary lull the patient may comply with a given direction. The well-meant attentions of the nurse and friends are misunderstood and generally actively resisted. Sleep is almost abolished; indeed, completely so in some cases during the entire attack. Food and medicine are administered with great difficulty. When the food is placed in the mouth the patient may spit it out, though in other cases it may be greedily swallowed. As the delirium reaches its height the mind becomes more and more confused, and the motor excitement manifests itself in senseless struggling or in purposeless and automatic movements, turning about the bed, aimless gestures, pushing, rubbing, etc.

The physical condition is indicative of great weakness, the color is pale, the surface of the body is cold and often moist, and the emaciation of the typhoid fever is rapidly and greatly accentuated. The pulse is small, sometimes slow, sometimes rapid; it is always weak. As a rule, abrasions and ecchymoses are observed on vari-

ous parts of the body. Generally they are the unavoidable results of the patient's struggles.

Acute delirium is a complication of short duration. It may last only a few hours; it never extends over more than a few days. Recovery is ushered in by the return of consciousness, which is generally quite rapid. The patient begins to recognize his surroundings and his hallucinations disappear. He begins to comply with the directions of the nurse, takes his food and, above all, begins to sleep. As a rule, the recovery is steady and uninterrupted; but at times it is broken in upon by recurrences of the delirium, generally transient in character. Recovery does not, however, always ensue. The exhaustion may proceed so far as to lead to stupor, and the patient may remain in this condition for a prolonged period of time. The final prognosis, however, of even this form of complication is relatively good. The great majority of cases of acute delirium following typhoid fever recover. However, emotional irritability and instability, hebetude, and physical weakness persist for several weeks after the delirium has ceased. The memory of the patient for the events of the attack is much obscured. He can seldom, if ever, give any but a vague account of his experiences.

A word of caution may not be out of place here in regard to the too free use of alcohol in the treatment of typhoid fever. The writer once saw in consultation a child in which the delirium proved not to be a sequel of the fever, but was really due to the large quantities of alcohol which had been administered. A marked and typical alcoholic multiple neuritis, sthenic in character and exquisitely painful, was also present.

2. **Confusional Insanity.** The second form of post-typhoid insanity to claim our attention is confusional insanity. Like the acute delirium following typhoid fever it closely resembles the confusion resulting from other infectious and exhausting diseases. It is characterized by obtusion, mental confusion, incoherence of ideas, illusions, hallucinations, and by a *prolonged* course. It is much more frequently met with as a sequel of typhoid fever than acute delirium. Typhoid fever most frequently induces exhaustion

gradually; it is only in exceptional cases in which this exhaustion comes on suddenly that acute delirium ensues. In by far the larger number of cases the more slowly acting causes induce the more gradual developing and more prolonged affection we are about to consider. In keeping with these statements the onset is much less rapid than in acute delirium. It does not make its appearance until some days after the fever has subsided; generally, however, within the first week. The patient becomes nervous, restless, and cannot sleep. Soon he becomes unaccountably afraid and excited, fears impending trouble or death, is obtuse, fails to comprehend readily, often complains that he cannot think, and he readily becomes confused. After several days the symptoms become so pronounced that the patient begins to lose the correct appreciation of his surroundings, or of the circumstances in which he is placed. He no longer knows where he is, mistakes the people about him for strangers, and often begs piteously to be taken home. To the illusions are soon added hallucinations. He hears threatening voices, shouts, and cries. He sees frightful objects or horrible looking men who load him with abuse and curses. As in acute delirium, the patient now believes that he is being injured, that serious bodily harm is about to be done him, that he is to be beaten, crushed, killed. In addition the illusions also play an important part, even greater than the hallucinations. The patient in his condition of fear is excessively watchful of his surroundings, which he constantly misinterprets. The commonest objects are misunderstood—a spoon is taken for a knife, a thermometer inspires deadly fear, a hypodermic injection is regarded as a savage onslaught with a dagger. The patient also catches words and phrases uttered by the bystanders with surprising readiness, always, of course, to misinterpret them. For this reason it is well not to whisper in the patient's presence, nor to make unnecessary gestures, nor to move about the room mysteriously.

Sometimes it is possible, by speaking distinctly and loudly, to attract the patient's attention for a short time. Feeding, when possible, can be accomplished by this means. The food should be

urged upon the patient by speech, by the proper presentation of food to vision and to the lips. Frequently, however, it is impossible for many hours at a time to bring the patient to himself or to a realization of his surroundings by any means whatever.

Although the hallucinations are most frequently of a terrifying and depressing character, they are not necessarily so. In rare instances they are pleasurable, and the patient may talk in a disconnected way about his wealth, the beauty and grandeur of his surroundings, and the glorious future that lies before him. Such expansive ideas also are now and then found in an intercurrent manner in the ordinary depressive form. In keeping with these facts the emotional state is usually one of depression and apprehension, infrequently one of slight exaltation. Laughing and singing are sometimes interspersed with the manifestations of fear, and at times slight eroticism is noticed.

The thoughts are disordered and tangled, while, as in the acute delirium, there is almost always some increase in the rapidity of the flow of ideas. Consciousness, as already stated, is much obtunded; frequently it is dream-like. More or less motor excitement is always present. It is, however, much less marked than in the delirium. The patient is restless, tries to get out of bed, tries to run about the room, struggles at times to get away, and may exhibit some tendency to violence. In some cases there is relative quiet from muscular weakness or, perhaps, from inhibition. In others the patient holds fast in a senseless sort of manner to surrounding objects or persons, or resists in a semi-passive way the attentions of the nurse. In other cases, again, he betrays evidences of automatism and tends to remain for some time in the position in which he has been placed. Symptoms such as these, however, are relatively infrequent.

The speech varies considerably. Sometimes whole sentences are uttered, at other times merely phrases, fragments, or incoherent and disjointed words. It is, however, much easier to gain some idea of the character of the delusions which pass through the patient's mind than in acute delirium; there they are largely a matter of inference, here they are often more or less plainly expressed. As

might be expected, sleep is much disturbed. Insomnia is always marked, especially at night. Food is taken badly, partly because it is not properly recognized and partly because of fear and the suspicion of poisoning; the latter idea has its groundwork largely in illusions and hallucinations of taste and smell.

The physical condition of the patient is, as a rule, bad. Loss of flesh is marked, though rarely as striking as in acute delirium. The surface is cool, the extremities often cold, sometimes moist. The temperature is not infrequently subnormal,[1] though it may be normal throughout. The pulse is slow and lacks force. Now and then there is incontinence. The reflexes, when they can be studied, are usually found exaggerated.

The symptoms attain a maximum in from two to three weeks after the actual onset. The subsequent course is apt to be irregular, the confusion becoming more or less marked by turns; the periods of temporary improvement often correspond to the taking of increased amounts of food, or follow more or less successful periods of sleep. Convalescence generally sets in very gradually. Generally many weeks elapse before persistent improvement is noted. The patient begins for short periods of time to properly appreciate his surroundings and to understand what is said to him. The periods of lucidity gradually become prolonged until, from being merely of a few hours' duration, they last through the greater part of the day. During the convalescence the patient is often irritable and hard to please. Sometimes traces of the old distrust and suspicion are seen; the patient makes absurd charges against his nurse, or is obstinate and intractable. Gradually, however, he becomes more sensible, more friendly, and begins to manifest confidence in those about him. In many instances, too, during this period, the patient is mildly excited or depressed, while in others some of the hallucinations persist after lucidity has made its appearance, but in such case the latter are no longer made the basis of delusions. Rarely, however, fleeting delusions now and then betray themselves. A valuable index as

[1] Wood. University Medical Magazine, Dec., 1889, vol. ii. p. 117.

to impending convalescence is the willingness of the patient to take food. Partial relapses, it should be added, also occur, especially as the result of emotional excitement, the visits of importunate and mistaken friends, or other imprudent management. The time occupied by the course of the disease varies from six weeks to four months, and sometimes longer. Even after recovery appears to have taken place the patient may betray decided mental weakness and readiness of fatigue. This asthenia is often prolonged, and may persist for months and, exceptionally, even for a year or more. Death as a result of typhoid confusional insanity is very infrequent. Death from suicide or accident should not be forgotten as a possibility.

Stuporous Insanity. Sometimes, though infrequently, cases which begin as confusional insanity merge into stupor, the nervous exhaustion becoming so profound that the mental faculties are finally completely suspended. However, cases that become stuporous differ from the ordinary confusional cases in the length of the developmental period, and although a stage of confusion is present preceding the onset of stupor, this stage is usually short. The stuporous form is, therefore, well defined clinically, but bears close relations to the form characterized by confusion.

Stuporous insanity is characterized chiefly by a more or less marked abeyance of the mental faculties. It is also known as acute dementia or curable dementia. It is of extremely gradual development. Several weeks usually elapse before stupor is established, and during this preliminary period the patient is nervous, timid, and fearful, sleeps badly, complains of headache, and is dull of comprehension. Instead of gaining in weight, as does the ordinary case of typhoid during convalescence from the fever, he is either at a standstill or loses. He is worried, feels ill, and loses his appetite. Soon mental confusion makes its appearance. As in confusional insanity the patient loses the proper appreciation of his surroundings. He believes himself to be away from home and fails to recognize the persons about him, and after a time this inability to interpret his surroundings gives way to an inability to appreciate them at all. The patient lies motionless in bed, indif-

ferent apparently to everything about him. In this condition he cannot be made to answer questions and does not speak spontaneously. Emotionally he seems placid and indifferent, though in some cases periods are present during which transient emotional movements, excitement, depression, or weeping are observed. The face is relaxed, flaccid, and expressionless. He is utterly helpless. Frequently he betrays a form of automatism; he may remain for some time in the position in which he has been placed without moving. Thus the arm may be kept elevated, the fingers extended, or the head turned to one side. These symptoms are often spoken of as cataleptoid, but they have, of course, no relation with true catalepsy. Again, while the great majority of cases are motionless, a very limited number are accompanied by agitation or purposeless movements. The feeding of the patient is often difficult. At times he will swallow food that is placed in his mouth, at other times he will allow it to remain in the mouth, making no effort at swallowing, or will allow it passively to escape upon the pillow. In many cases nasal feeding is the only practicable plan of administering nourishment, and, as a rule, this can easily be carried out and answers every possible purpose.

The physical condition of the patient reveals great depression of nutrition. There is decided loss of flesh, coolness or coldness of the surface and at times a subnormal temperature. The features are pale, perhaps slightly cyanosed. The extremities are often bluish and sometimes œdematous. The pulse is small and slow, the respiration shallow. In women the menses cease. Like confusional insanity, stupor is an affection of long duration; several months are always required. Convalescence also is established very gradually. The patient begins by betraying some consciousness of his surroundings. He may attempt to speak or make movements of expression. He also begins to take his food more readily, brightens up a little toward the latter part of the day, and little by little comes into normal relations with his environment. Readiness of fatigue persists for a long time, and there are frequent recurrences of mental confusion which reveal themselves either in the patient's actions or in his conversation. Great care

should be taken to conserve the strength of the patient as much as possible by the avoidance of excitement or of visitors. While by far the greater number of cases end in recovery, this is not the invariable rule. A few cases pass into permanent dementia; in others some permanent mental impairment persists, and in a smaller number death results, due either to the gravity of the exhaustion or to some visceral complication.

3. **Cerebral Asthenia, Pseudo-dementia, Pseudo-paresis.** More frequently, perhaps, than any other complication we have following typhoid fever a condition of general mental enfeeblement. This is generally of short duration, but is sometimes excessively prolonged. There is present in such cases a slight, though unmistakable, weakness of the intelligence together with abnormal excitability and loss or impairment of emotional control. The patient does not comprehend as readily as normally, is incapable of sustained effort, lacks spontaneity of thought, and laughs or cries on relatively slight provocation. He is also very readily fatigued. At times there is in addition a diminution in the facility and readiness of speech. Physical symptoms indicative of weakness are also present—*e. g.*, coldness of the extremities, cardiac palpitation, atonic indigestion and persistent sleep disturbances. This cerebral asthenia for some unexplained reason, occasionally follows comparatively mild attacks of the fever and may be very marked. In other cases, again, in which the attack has apparently been of great severity, these symptoms may be entirely absent.

Instead of a mere mental weakness and anenergia, actual mental obtusion may be present, and this mental obtusion may become so pronounced as to lead to great impairment of all of the mental faculties—a form of dementia. This is not, however, a true dementia, but one in which the mental faculties are merely suspended, not obliterated. It is properly termed a pseudo-dementia. This pseudo-dementia lasts many months and at times even one or two years. Recovery follows in the majority of cases, but is very gradual. Sometimes it is incomplete, permanent mental impairment resulting. Every now and then there are added to this

background of dementia symptoms which closely resemble those of paresis. Thus there may be present great muscular weakness, ataxia of movement, tremor of the lips, face, or extremities,[1] and to the condition of obtusion, hebetude, and mental weakness already present, there may be added absurd and ambitious delusions. This feeble, expansive state makes the resemblance to paresis appear very striking and often misleading. The pseudo-paresis of typhoid fever may occasion difficulty in diagnosis if the physician be in ignorance of the etiology. However, the detailed history of the case, the presence or absence of the Argyll-Robertson pupil, the condition of the optic nerve as revealed by the ophthalmoscope, are among the factors which should be considered. Pseudo-paresis following typhoid fever almost always terminates in recovery; besides the course of the disease is different from that of paresis. The mental loss, too, is not as profound or as real.

4. **Insanity with Systematized Delusions Resembling Paranoia.** A very limited number of cases of insanity following typhoid fever present a series of more or less well-systematized delusions. These delusions are at times remains of the fever delirium which have persisted. At other times they arise during convalescence. The patient may give well-connected accounts of frightful persecutions, of murders, hangings, etc. The delusions are almost invariably of a depressive character, and appear to be connected with painful or terrifying hallucinations. Such cases have been described by Müller,[2] Hurd,[3] and others. They are distinguished from true paranoia not only by the peculiar etiology, but also by the fact that the delusions are not firmly fixed, but often shifting in character, and also by the fact that sooner or later, as soon as the general condition of the patient improves, the delusions vanish. Recovery may, however, not always ensue, and progressive mental impairment, with final dementia, may be the result. Such an outcome, however, appears to be exceptional.

5. **True Mania or True Melancholia.** In addition to the various forms of mental disorder above described, and which are

[1] Christian, Westphal, Regis.

[2] Müller, Loc. cit.

[3] Hurd, American Journal of Insanity, July, 1892.

evidently associated with the excessive nervous weakness and, perhaps, the profound intoxication of the typhoid infection, pure insanities are every now and then observed. In other words, true mania or true melancholia may arise subsequent to typhoid fever. Owing to the loose way in which the terms mania and melancholia are employed by many medical writers, many cases of so-called mania and melancholia have been placed upon record as resulting from typhoid fever. A close examination, however, reveals that they are in most instances cases of an insanity of exhaustion, generally confusional insanity, which have been classed as mania or melancholia, according to the presence of mental excitement on the one hand or mental depression on the other. Pure mania or pure melancholia, as a result of typical typhoid fever is excessively rare. For instance, typical melancholia with excessive psychic pain and self-accusatory delusions, as typified by the delusion of the unpardonable sin, is almost never met with. This is also true of pure mania as typified by excessive exaltation, expansion, and increased rapidity in the flow of ideas, without hallucinations or confusion. Further, cases of the pure insanities following typhoid fever do not, as a rule, like the insanities of exhaustion, develop immediately after or within a short period of the defervescence of the fever, but at rather later periods—weeks and months afterward. It is exceedingly probable that when a pure insanity does follow typhoid fever it is an *indirect* sequel. In other words, the post-typhoid condition of asthenia merely offers a suitable soil in which true mania or true melancholia may develop in subjects predisposed to these affections by heredity. We should remember that mania and melancholia are largely determined by heredity and only need a condition of depraved nervous nutrition in order to make themselves manifest.

Prognosis in General. The prognosis of the various mental complications of typhoid fever depends largely upon the period at which the symptoms appear. Prodromal insanity, especially grave prodromal delirium, tends in a large number of cases, one-third, according to Adler, to end fatally. The prognosis of the complications arising during the fever is almost uniformly good. The

fever-delirium, the confusion, the expansive and ambitious ideas vanish with the disappearance of the fever. The various forms of mental derangement which occur as sequelæ of typhoid fever also offer a favorable prognosis as a whole. The great majority of cases of post-typhoid confusional or stuporous insanity make a good recovery, but this is not by any means the constant result. Instead of a continuous progress toward recovery, there may be a series of relapses, followed by incomplete recovery or cases may pass into hopeless chronicity and dementia. This, however, as has already been pointed out, is the outcome in a small percentage of cases only. Pilgrim[1] states that in his opinion only about 50 per cent. of cases due to typhoid fever recover, while 20 per cent. die from exhaustion, and 30 per cent. gravitate into chronic insanity. These statements, however, are not borne out by the experience outside of the asylums. The percentage of favorable results is really much greater.

It may be not uninteresting to add a paragraph as to the remarkable effects which follow typhoid fever when attacking those who are already insane. In quite a number of such cases, irrespective of the special form of insanity, recovery follows typhoid fever. In others, again, long-continued improvement ensues; in a smaller number temporary improvement, and in others still no change whatever is observed. Nasse,[2] Wise,[3] Keay,[4] Charon,[5] and others have placed on record quite a number of cases of recovery.[6] The interesting fact of recovery of insanity after typhoid fever is comparable to the effects of other infectious processes, such as erysipelas, and also to the results occasionaly following trauma and surgical operations on the insane. Even in so grave a mental

[1] State Hospital Bulletin, New York, Utica, 1896, vol. i. p. 50.

[2] Loc. cit.

[3] State Hospital Bulletin, New York, Utica, 1896, vol. i. p. 63.

[4] Journal of Mental Sciences, 1896, vol. xlii. p. 267.

[5] Charon, Arch. de Neurol., 1896, i. p. 330.

[6] Hyvert, Arch. de Neurol., 1895, vi. p. 103, believes on the other hand, that typhoid fever affects the mental state of the insane to a less degree than do other infections.

disease as paresis, an attack of erysipelas or a trauma is occasionally followed by a striking and remarkable remission of symptoms; similar statements may be made with regard to melancholia and other forms of mental disease associated with depression and impaired nutrition. In cases in which typhoid fever fails to cure or to improve the mental symptoms, the psychosis already present does not appear to be affected injuriously. At least this is Nasse's[1] conclusion. One case under the observation of this writer presented a paroxysm of delirium of short duration; in none of the others, five in number, in which the typhoid infection failed to cure the insanity, did any unfavorable result supervene. Nasse[1] further observed a greater percentage of recoveries from typhoid fever in the insane than among the hospital attendants. Wise,[2] on the other hand, found the mortality 30 per cent. among the insane and 24 per cent. among the employes. These data evidently do not point to any lessened degree of vulnerability on the part of the insane.

[1] Hyvert, Arch. de Neurol., 1895, vi. p 103.

[2] State Hospital Bulletin, New York, Utica, vol. i. p. 69.

INDEX.

ABDOMINAL, catarrh, 70
pain, 123
Abnormal eruptions in typhoid fever, 166
Abortive typhoid fever, 38, 73
Abrupt onset, 56
Abscess, alveolar, 205
appendicular, 148
of liver, 209
of lung, 97, 182
of mesentery glands, 218
of parotid gland, 208
subphrenic, 218
of retroperitoneal glands, 218
subdiaphragmatic, 218
Absence of fever, 70
of intestinal lesions, 115
Acute, delirium, 267, 269
endocarditis, 100
nephritis, 104
pleurisy in onset, 51
pulmonary tubercular consolidation, 50
Afebrile abdominal catarrh, 70
Age, mortality in advanced, 36
Albuminuria, 104
Alcoholic neuritis, 225
Alimentary canal in convalescence, 202
in developed stage, 109
in onset, 53
Alveolar abscess, 205
Amaurosis, 235
Ambiguous period, 64
Anus, gangrene of, 208
Aphasia, 162, 234
Aphthous inflammations, 205
Appendicitis, 143, 257
Apyretic typhoid fever, 71
Ascending myelitis, 228
Asthenia, 101

BED, fever, 181
sores, 169
Bilious fever, 54
Blood, bacillus of Eberth in, 103
in developed stage, 101
Bloody stools, 78
Boils, 241
Bowel, cicatricial contraction of, 204
gangrene of, 203
thermometer in, 221
Bronchitis, 95

CARBUNCLES, 241
Carphologia, 164
Catarrh, afebrile abdominal, 70
Cerebral, asthenia, 277
embolism, 160
thrombosis, 160
Changing fortunes, period of, 64
Children, relapse in, 177
typhoid fever in, 27
Chill in onset, 43
Chills, 75
Cholangitis, 130
Cholecystitis, 42, 89, 211
Chorea, 237
Cicatricial constriction of bowel, 204
Circulation in convalescent stage, 182
in developed stage, 98
Coal-tar products, chill producing, 78
Collapse, 78, 79, 100, 127
Complicating infections, 78
Complications, mental, 265
Conditions which ape typhoid fever, 253
Confusional insanity, 271
Congestion, hypostatic, 96
Constipation, 120, 202
Convalescence, period of, 174
Convalescent stage, circulation in, 182
respiratory affections in, 181
Convulsions, 162
Course of fever in relation to prognosis, 83
Croupous pneumonia, 67, 96
Cystitis, 109

DEATH, sudden, 184, 188
Delirium in developed stage, 149
in onset, 57
Desquamation of skin, 248
Developed stage, 63
alimentary canal in, 109
blood in, 101
circulation in, 98
respiratory system in, 85
Diagnosis of perforation, 129
Diarrhœa, 80, 202
in typhoid fever, 120
serous, 55
Dicrotism, 98
Diphtheria of intestine, 203
Dislocation of joints, 251
Duration, 262

EMBOLISM, 185
cerebral, 160
Emphysema of mediastinum, 93
Empyema, 98
Endarteritis, 187
Endocarditis, 185, 186
acute, 100
ulcerated, 80
Eosinophiles, 80
Epididymitis, 194
Epilepsy in relation to typhoid fever, 165
Epistaxis, 88
late, 89
Eruptions, abnormal, 166
Eruptive diseases in the course of fever, 171
Erysipelas, 241
Exhaustion, 101

FAINTNESS, 78
Fall of temperature, sudden, 78
Fever, bilious, 54
gastric, 54
infantile remittent, 54
pneumo-typhoid, 47
remittent malarial, 74
septic, 75
worm, 54
Fœtus, infection by typhoid fever, 33
Forme cardiaque, 99
Frequency of hemorrhages, 123
of typhoid fever in children, 27

GALL-BLADDER, 80
Gallstones, 213
Gangrene, 170
of anus, 208
of bowel, 203
of extremities, 192
of lung, 97, 98, 182
of mouth, 205
of perineum, 208
of skin, 241
of tongue, 206
superficial, 169
Gastric fever, 54
symptoms in onset, 54
Genito-urinary complications of convalescence, 193
tract in developed stage, 104
Glossitis, 205
Green stools, 121

HÆMATURIA, 52, 104
Headache, 152
Heart muscle in typhoid fever, 183
Hebetude, 150
Hemiplegia, 229
Hemoptysis, 97
Hemorrhages, 123
frequency of, 123
from stomach, 111
in children, 125
intestinal, 78
Hemorrhagic diathesis, 170
eruptions, 170
infarction of lungs, 96
nephritis, 104
Hemorrhoidal arteries, thrombosis of, 207
Hernia, scrotal, 132
ventral, 132
Herpes labialis, 170
High temperatures, 66
Hyperpyrexia, 66
Hypostatic congestion of lungs, 96
Hysteria, 163, 238
Hysterical convulsions, 152

IMMUNITY, 262
Indigestion, 202
Infantile remittent fever, 54
Infarction, 185
of lung, 96
Infections, complicating, 78
Influenza, 79
Insanity, 152, 268
Insidious perforation, 128
Intercurrent relapse, 67, 177
Intestinal hemorrhage, 78
lesions, absence of, 115
Intestines in typhoid fever, 115

JAUNDICE, 219
Joints, 250

KIDNEY, miliary abscess of, 107
Kidneys, in onset of fever, 52
Knee-jerks, absence of, 164

LAPAROTOMY in perforation, 135
Laryngeal form of typhoid, 51
paralysis, 235
ulceration, 90
Laryngo-typhus, 91
typhoid, 51
Larynx, necrosis of, 94
perichondritis of, 89
Late epistaxis, 89
Leucocytes in complications, 102
Linea albicantes, 249
Liver, abscess of, 209
Lobular pneumonia, 95
Localized atrophy of skin, 249

Lung, abscess of, 182
gangrene of, 182
Lungs, hemorrhagic infarction of, 96
hypostatic congestion of, 96
tuberculosis of, 79

MAIDSTONE epidemic, 121
Malaria, 253
Malarial fever, remittent, 74
Maniacal delirium, 58
Measles, complicating, 173
Mediastinal emphysema, 93
Mediastinum, suppuration of, 93
Melancholia, 278
Meningitis, 153
Mental complications, 265
disturbances, 234
Mesentery glands, abscess of, 218
Miliary abscess of kidney, 107
tuberculosis, 96, 256
Morbidity, decrease of, 18
Mortality, decrease of, 18
of perforation, 130
relation of age to, 36
Mountain fever, 260
Mouth, gangrene of, 205
Multiple abscess of liver, 210
relapses, 180
Myelitis, ascending, 228
Myostitis, 237

NECROSIS of larynx, 94
Nephritis, acute, 104
hemorrhagic, 104
Nephro-typhoid, 52
Nervous symptoms in convalescence, 221
in developed stage, 149
in onset, 56
Neuritis, 165
peripheral, 221
Noma, 204

ŒDEMA of thorax, 249
Œsophagus, inflammation of, 110
ulceration of, 110, 206
Onset, 37
abrupt, 56
chill in, 43
delirium in, 57
in alimentary canal, 53
in kidneys, 52
nervous symptoms in, 56
respiratory infection in, 47
rigor in, 43
skin in, 62
temperature in, 37
unusual temperature variations in, 40
Orchitis, 193

PAIN, abdominal, 123
Paralysis, 165, 221
agitans, 235
laryngeal, 235
of vocal chords, 235
pseudo-hypertrophic, 238
Paranoia, 278
Parotid gland, abscess of, 208
Parotitis, 209
Percentage of perforation, 130
Perforation, 78, 126
diagnosis of, 129
of bowel, 202
treatment of, 135
Pericarditis, 188
Perichondritis of larynx, 89
Perineum, gangrene of, 208
Period, ambiguous, 64
of convalescence, 174
of "steep curves," 64
Peripheral neuritis, 221
Peritoneum, tuberculosis of, 79
Peritonitis, 129, 204
Pharyngeal involvement in developed stage, 109
Pharyngo-typhoid, 53
Phlebitis, 67, 191
of calf of leg, 193
Pleurisy, 67, 97
acute, in onset, 51
Pneumonia, croupous, 96
lobular, 95
Pneumothorax, 97
Pneumo-typhoid fever, 47
Pregnancy complicating typhoid fever, 32, 64
typhoid fever during, 32
Profuse urinary flow, 109
Prognosis, course of fever in relation to, 83
in post-typhoid insanity, 279
Pseudo-dementia, 277
hypertrophic paralysis, 238
paresis, 277
Puerperal septicæmia, 259
Pulmonary abscess, 97
tubercular consolidation, 50
Pulse rate, 98
Pyelitis, 107
Pyemia, 80
Pylephlebitis, 211
Pyonephrosis, 109
Pyuria, 107

RAPID pulse, 99
Recrudescence, 174
Relapse, 174
from gall-bladder infection, 214
in children, 177
intercurrent, 67, 177

Relation of age to mortality, 36
Remittent malarial fever, 74
typhoid fever, 74
Respiratory affections in convalescent stage, 181
infection in onset, 47
system in developed stage of disease, 85
Retention of urine, 53
Retroperitoneal glands, abscess of, 218
Rigor, 75, 181
in onset, 43
Rose rash, 80, 165

SCARLET fever, 248
complicating typhoid, 171
Scrotal hernia in typhoid fever, 132
Second attacks, 262
Septic fever, 75
Septicæmia, 80
Skin in convalescence, 241
localized atrophy of, 249
in onset, 62
in well-developed stage, 165
Spleen, affections of, 103
enlargement of, 220
rupture of, 220
Spleno-typhoid, 103
"Steep curves," 64
Stomach, 110
typhoid ulcers of, 111
hemorrhage of, 111
Stools, bloody, 78
green. 121
Strabismus, 235
Stupor, 268
Stuporous insanity, 275
Submaxillary glands involved, 110
Subphrenic abscess, 218
Subsultus tendinum, 164
Sudamina, 168
Sudden death, 184, 188
fall of temperature, 78
rises in temperature, 57
Sudoral typhoid fever, 44
Suffocative attacks, 92
Suppuration of mediastinum, 93
Syphilis, 80
Systematized delusions, 278

TACHE bleuâtre, 73, 169
cérébrale, 168
Temperature, absence of febrile, 70
in convalescence, 174
in developed disease, 63
in onset, 37
in children, 42
rises in, 67
sudden fall of, 78
variations from usual in onset, 40
Tetany, 238
Thermometer in bowel, 221
Thorax, œdema of, 249
Thrombosis, 182
cerebral, 160
of hemorrhoidal arteries, 207
Thyroid gland, 249
Tongue, gangrene of, 206
Treatment of perforation, 135
Tremor, 235
Trichinosis, 80
True mania, 278
Tubercular consolidation, 50
Tuberculosis, 95, 185, 256
of lungs, 79
of peritoneum, 79
Typhoid bacilli in urine, 108
fever, abortive, 38, 73
apyretic, 71
change in type of, 27
conditions resembling, 253
constipation in, 120
diarrhœa in, 120
eruptions, 166
general considerations of, 17
in children, 27
in pregnancy, 32
infection of fetus by, 33
intestines in, 115
laryngeal form, 51
low temperatures in, 70
remittent, 74
sudoral, 44
"Typhoid-spine," 238
Typho-malarial fever, 81
Typhus, laryngo, 91
levissimus, 73

ULCER of stomach, 111
Ulcerative endocarditis, 80
Ulceration and diarrhœa, relation of, 121
of larynx, 90
œsophageal, 110, 206
Urine, retention of, 53
typhoid bacilli in, 108
Urticaria, 168

VARICOSITY of subcutaneous veins 192
Varieties of onset, 37
Veins, varicosity of, 192
Ventral hernia in typhoid fever, 132
Violent diarrhœa in onset, 55
Vocal cords, paralysis of, 235
Vomiting, 55, 111

WORM fever, 54

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